"A PROSPECTIVE, RANDOMIZED COMPARATIVE STUDY ON EFFECT OF ADDING CLONIDINE vs DEXMEDETOMIDINE TO EPIDURAL BUPIVACAINE (0.125%) ON POSTOPERATIVE ANALGESIA IN UPPER ABDOMINAL SURGERIES"

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CERTIFICATE

This is to certify that the dissertation entitled, **"**A PROSPECTIVE, RANDOMIZED COMPARATIVE STUDY ON EFFECT OF ADDING CLONIDINE vs DEXMEDETOMIDINE TO EPIDURAL BUPIVACAINE (0.125%) ON POSTOPERATIVE ANALGESIA IN UPPER ABDOMINAL SURGERIES" submitted by Dr. SWARNALINGAM.T in partial fulfilment for the award of the degree of Doctor of Medicine in Anaesthesiology by the Tamilnadu Dr. M.G.R. Medical University, Chennai is bonafide record of the work done by him in the INSTITUTE OF ANAESTHESIOLOGY & CRITICAL CARE, Madras Medical College, during the academic year 2009-2012.

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CONTENTS

INTRODUCTION

Effective pain control is essential for optimal care of surgical patients. Adequate post-operative pain relief must be an integral part of administration of anesthesia.Inadequate post-operative pain relief may result in clinical and psychological changes that may increase the morbidity and mortality as well as the cost of treatment as a whole, in addition to decreasing the quality of life post –operatively.

The use of epidural analgesia for the management of post operative pain has evolved as a critical component of multi modal approach to achieve the role of adequate analgesia with improved outcome. Epidural analgesia offers superior post operative pain relief compared with systemic drugs. In addition to improved pain control, epidural analgesia can improve patient outcome by attenuating detrimental post operative stress.[20,21]

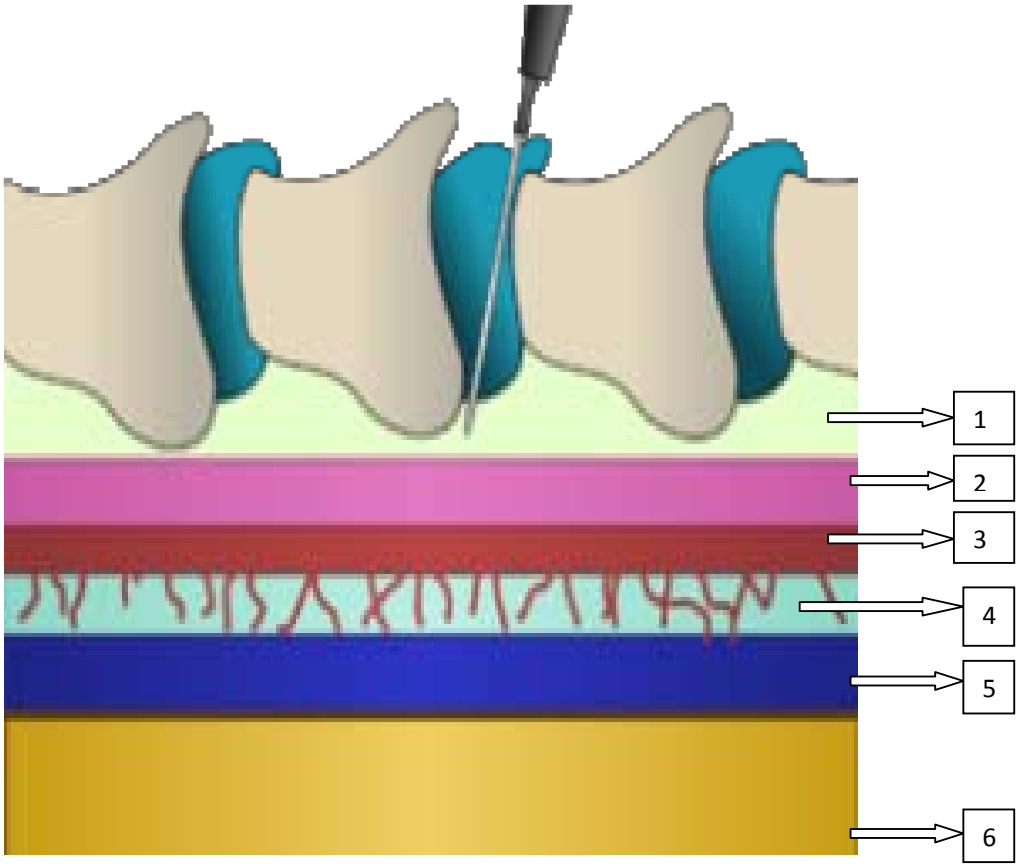
Sedation, stable hemodynamics and an ability to provide smooth and prolonged post operative analgesia are the main desirable qualities of an adjuvant in post operative epidural analgesia .α-2 adrenergic agonists have both analgesic and sedative properties when used as an adjuvant in regional anaesthesia [2-6]. Dexmedetomidine is a highly selective α-2 adrenergic agonist with an affinity of eight times greater than clonidine. The analgesic requirement gets reduced to a huge extent by the use of these two adjuvants because of their

analgesic properties and augmentation of local anaesthetic effects as they cause hyperpolarisation of nerve tissues by altering transmembrane potential and ion conductance at locus coerulues in the brain stem.[11, 17, 18, 23, 24] The stable hemodynamics and the decreased oxygen demand due to enhanced sympathoadrenal stability make them very useful pharmacologic agents.[36]

Keeping their pharmacologic interactions and other properties we planned to conduct a single blinded prospective randomized clinical controlled study at our institution in patients who underwent upper abdominal surgery under general anaesthesia with an aim to compare the analgesic and sedative effect of these drugs given via epidural route as an adjuvant to 0.125% bupivacaine in the post operative period.

ANATOMY OF EPIDURAL SPACE

The epidural space is smaller than the subarachnoid space.It extends from the base of the skull to the sacral hiatus and surrounds the dura mater anteriorly, laterally, and posteriorly. The epidural space is bound posteriorly by the ligamentum flavum and laterally by the pedicles and the intervertebral foramina.



1- Epidural space 2- Duramater 3- Arachnoid mater 4- Subarachnoid space 5-Piamater 6- Spinal Cord

The **epidural space**, that exists between it and the internal surfaces of the vertebral bones and their supporting ligamentous structures. This space is likewise pressed closed by surrounding tissue pressure, so it is called a 'potential' space.

Contents of epidural space:

It is a space filled with the fat, areolar tissue, lymphatics, veins, and nerve roots that traverse it, but no free fluid. The volume of fat is greater in obese individuals and less in the elderly. It is postulated that the decrease in epidural fat explains the age-related changes in epidural dose requirements. The epidural space is rich in blood vessels, including Batson's venous plexus. Batson's plexus is continuous with the iliac vessels in the pelvis and the azygos system in the abdominal and thoracic body walls . Because this plexus has no valves, blood from any of the connected systems can flow into the epidural vessels. This is especially important in obstetrics when compressed caval vessels can lead to engorgement of the epidural veins, increasing the risk of catheter entry into a vein. The engorgement is even greater at the intervertebral foramina where the vessels egress from the vertebral canal.

Physiological effects of epidural blockade

The primary site of action of local anaesthetic solutions injected into the epidural space is the spinal nerve roots. The segmental nerve roots in the thoracic and lumbar regions are mixed nerves, containing somatic sensory, motor, and autonomic nerve fibres. Sensory blockade interrupts the transmission of both somatic and visceral painful stimuli, whereas motor blockade provides muscle relaxation with a varying degree of sympathetic blockade. The injection site for epidural anaesthesia should be close to the nerve roots of interest in order to obtain the best results with minimal amount of local anaesthetic and decreased risk of side effects from systemic absorption of the local anaesthetic (catheter/incision congruent). Differential nerve block, an important concept for epidural anaesthesia, refers to the phenomenon in which nerve fibres with different functions demonstrate a varying sensitivity to the effects of local anaesthetics. Sympathetic fibres are usually blocked first followed by pain/temperature, then proprioception, followed by motor blockade. After an epidural block, sympathetic blockade (temperature) may vary from zero to four segments higher than the sensory block level (pain/light touch), which is two segments higher than motor blockade. Regression of the block occurs in reverse order. The physiologic effects of epidural blockade on organ systems depends on the spinal level and the number of spinal segments blocked. In general, high thoracic epidural blocks and extensive epidural blocks

are associated with more profound sympathetic block, resulting in a more profound physiologic effect in the cardiovascular system.

Pharmacology of epidural blockade

To be successful with epidural blockade, the clinician must understand the physiology of nerve conduction and the pharmacology of the local anaesthetics. Potency and duration of the drugs, their ability to preferentially block sensory and motor fibres, as well as the anticipated duration of surgery or need for postoperative analgesia are factors to be considered before instituting epidural blockade. The principal site of action of local anaesthetics after epidural injection is thought to be the spinal nerve roots, the spinal cord, and possibly the brain. Nerve fibres with different features and function display varying sensitivity to local anaesthetic blockade. For example, sympathetic fibres (thin, myelinated when entering the sympathetic trunk) tend to be blocked with the lowest concentration of drug, followed by pain, touch, and finally motor fibres.

Action of local anaesthetics

Local anaesthetic binds to sodium channels, primarily in the inactivated state, preventing further channel activation. Sodium ion movement into the cell

is prevented, effectively blocking the development of the action potential. The resulting resting membrane potential is unaffected by further nerve stimulation, referred to as membrane stabilization of local anesthetics.

Mechanism of action of local anaesthetics in neural blockade

Within the dorsal horn, local anaesthetics can block both sodium and potassium ion channels in the dorsal horn neurons, inhibiting the generation and propagation of pain signals (nociceptive electrical activity). Motor blockade occurs from a similar action on the ventral horn neurons. Blockade of calcium ion channels in the spinal cord leads to resistance of electrical stimulation from nociceptive afferent nerves, creating an intense analgesic action seen in centrally administered local anaesthetics. In addition to ion channel alterations in the central neuraxis, epidurally administered local anaesthetics indirectly inhibit the release of substance P and other neurotransmitters involved in pain signal processing. Substance P is involved in pain transmission from the presynaptic terminals of dorsal root ganglionic cells. The putative effect of centrally administered local anaesthetics on substance P and these other transmitters is linked to the presynaptic blockade of the voltage-gated calcium channel. When calcium entry is blocked at the presynaptic level, release of these neurotransmitters (glutamate, substance P, calcitonin gene-related peptide [CGRP], neurokini-1 and -2 [NK1, NK2]) at the presynaptic level does not

occur. Therefore, epidurally administered local anaesthetics can indirectly inhibit pain signal transmission.

Indications:

Injecting medication into the epidural space is primarily performed for analgesia. Epidural analgesia may be used:

1. For **analgesia alone**, where surgery is not contemplated. An epidural for pain relief (e.g. in childbirth) is unlikely to cause loss of muscle power, but is not usually sufficient for surgery.

2. As an **adjunct to general anaesthesia**. The anaesthetist may use epidural analgesia in addition to general anaesthesia. This may reduce the patient's requirement for opioid analgesics. This is suitable for a wide variety of surgery, for example gynaecological surgery (e.g. hysterectomy), orthopaedic surgery (e.g. hip replacement), general surgery (e.g. laparotomy) and vascular surgery (e.g. open aortic aneurysm repair).

3. As a **sole technique for surgical anaesthesia**. Some operations, most frequently Caesarean section, may be performed using an epidural anaesthetic as the sole technique. Typically the patient would remain awake during the operation. The dose required for anaesthesia is much higher than that required for analgesia.

4. For **post-operative analgesia**, after an operation where the epidural was used as either the sole anaesthetic, or was used in combination with general anaesthesia. Analgesics are given into the epidural space for a few days after surgery, provided a catheter has been inserted. Through the use of a patient-controlled epidural analgesia (PCEA) infusion pump, a patient has the ability to give an occasional extra dose of post-surgical pain medications administered through the epidural.

5. For the **treatment of back pain**. Injection of analgesics and steroids into the epidural space may improve some forms of back pain.

6. For the **treatment of chronic pain** or **palliation of symptoms** in terminal care.

POST OPERATIVE EPIDUARL ANALGESIA

The pain experienced after a surgery involving the abdominal musculature leads to a reduction in vital capacity of 70-75% in upper abdominal and 50% in lower abdominal surgeries. Thoracic epidural analgesia provides not only complete freedom from pain, but also a very substantial improvement in the vital capacity. Search for an ideal adjuvant for post operative epidural analgesia still continues that could result in reliable prolongation of post operative pain relief without side effects.

Post operative pain:

It has deleterious effects on every system of the body.

Cardiovascular	Tachycardia, hypertension, increased SVR, increased cardiac work.
Pulmonary	Hypoxia, hypercarbia, atelectasis, decreased cough, decreased VC, FRC, V/Q mismatch.
Gastrointestinal	Nausea, vomiting, ileus.
Renal	Oliguria, urinary retention.
Extremities	Skeletal muscle pain, limited mobility, thrombo embolism.

Endocrine	Increased adrenergic activity, increased metabolism, increased oxygen consumption.
CNS	Anxiety, fear, sedation, fatique
Immunologic Impairment	

BENEFITS OF POST OPERATIVE EPIDURAL ANALGESIA

Cerebral	Improved post-OP cognition, reduction in CVA
Cardiovascular	Reduced MI, reduced blood loss, reduced transfusion requirement, reduced DVT
Pulmonary	Reduced pulmonary infection, reduced pulmonary embolism, reduced respiratory depression, reduced hypoxemia
Stress Response	Reduced stress catecholamines
Gastrointestinal	Reduced ileus
Renal	Reduced ARF
Surgical outcomes	Reduced length of stay, reduced morbidity & mortality

Complications of epidural analgesia:

1. Subdural puncture

The patient should be assessed for a sudden or progressive increase in side effects such as sedation, loss of sensory and motor function, hypotension.

2. Epidural abscess

The catheter insertion site should be assessed every 8 hours for signs of infection i.e. tenderness, erythema, swelling, drainage. And also for changes in sensory/motor function every 4 hours including unexplained back pain, bowel or bladder dysfunction, fever or neck stiffness.

3. Epidural haematoma

The catheter insertion site must be assessed every 8 hours for pain and or swelling at the site and also for changes in sensory/motor function for every 4 hours including progressive numbness, weakness or bowel and bladder dysfunction.

4. Migration of catheter into epidural vessels

The catheter may migrate into the blood vessels of the epidural space causing the medications to be delivered systemically.

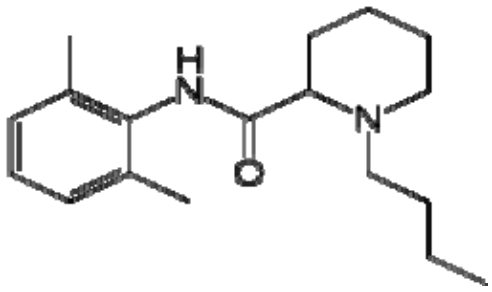
PHARMACOLOGY OF BUPIVACAINE

Bupivacaine is an amide local anaesthetic, synthesized by A.F.Ekenstam in 1957 and brought into clinical use in 1963.

It is produced for clinical use in a racemic mixture, containing equal proportions of the 'S' and 'R' enantiomers. It is supplied for clinical use as a hydrochloride salt.

Chemical Structure

Description: ± 1- Butyl-N-(2, 6-dimethylphenyl) – 2- piperidine Decarboxamide Hydrochloride monohydrate.



Physico-chemical Profile

Molecular weight (base)	-288
pKa	-8.1
Lipid Solubility	-28
Plasma Protein Binding	-95%

Mechanism of Action

Bupivacaine exerts its effects by inhibition of sodium channels. It acts to block conduction in the nerves by decreasing or preventing the large transient increases in permeability of the cell membrane to sodium ions that follows depolarization of the membrane. Bupivacaine also reduces the permeability of the resting nerve membrane to potassium as well as sodium ions.

Pharmacodynamics

Bupivacaine by virtue its pharmacological effects, has a stabilizing action on all excitable membranes. In the central nervous system, stimulation can occur producing restlessness, tremors and convulsions in over dosage. Bupivacaine also causes a reduction in the automaticity of the heart.

The clinical profile of nerve blockade produced by Bupivacaine differs from that of lignocaine. It is 4 times more potent than lignocaine, but the onset of action is slower. The duration of action is considerably longer. The sensory block produced by Bupivacaine tends to be more marked than the motor block.

Pharmacokinetics

Bupivacaine is rapidly absorbed from the site of injection. The rate of rise in plasma Bupivacaine concentration and the peak plasma concentrations obtained depend on the route of administration. There is also some inter

individual variation and peak systemic concentrations may occur between 5 and 30 minute after administration. The addition of vasoconstrictor delays absorption and results in lower plasma concentration of Bupivacaine.

Pharmacokinetic Profile

Volume of distribution at steady rate (Vdss)	72 lrs
Clearance	0.47 L.min
t½α	2.7 min
t½β	28 min
t½γ	3.5 hrs

Metabolism

Possible pathways for metabolism of Bupivacaine include aromatic hydroxylation, N-dealkylation, amide hydrolysis and conjugation. Only the N-dealkylated metabolic N-esmethylbupivacaine has been measured in blood and urine after epidural and spinal administration. The degradation of Bupivacaine takes place in the liver. Renal disease is unlikely to alter the kinetics of Bupivacaine to any great extent. Less than 10% of the drug is excreted unchanged in urine.

The onset of action of Bupivacaine occurs 20-30 minutes after a peripheral nerve block and duration lasts for 8-9 hours.

Clinical Applications

Infiltration anaesthesia

Peripheral nerve blocks

Central neruraxial blocks (intrathecal, epidural and caudal)

Contradictions

Para cervical block

Known hypersensitivity to amide local anaesthetics

Intravenous regional anaesthesia (IVRA)

Preparations available

0.25%, 0.5% solutions in 10 ml and 20 ml vials.

5mg/ml (0.5%) bupivacaine and 80 mg dextrose in 4 ml ampoules for intrathecal injection (Baricity 1.0207)

Recommended Safe Dose

Concentration Used	Maximum Permitted Dose
0.125%-0.5%	3mg/kg body weight
0.75% (not to be used in obstetric epidurals)	Max.over 4 hrs-150mg Max. During 24 hrs-400 mg
0.5% plain/hyperbaric solution (intrathecal use)	20 mg

Adverse Reactions

Adverse reactions are associated mainly with excess plasma levels of the drug, which may due to over dosage, unintentional intravascular injection or slow metabolic degradation.

CNS Reactions

Excitation characterized by restlessness, anxiety, dizziness, tinnitus blurred vision or tremors possibly proceeding to convulsions, followed by drowsiness, unconsciousness and cardiac arrest.

Cardiovascular System Effects

Part of the cardiac toxicity that occurs from high plasma concentrations of Bupivacaine occurs because of blockade of cardiac sodium channels. Accidental intravenous injection of Bupivacaine causes cardiac dysarrthymias, atrioventricular block, verticular tachycardia and ventricular fibrillation. Pregnancy increases the sensitivity of cardio toxic effects of Bupivacaine.

Allergic Reactions

Manifests as urticaria, pruritus, angioneurotic edema etc. Cross sensitivity among members of amide type local anaesthetics has been reported.

PHARMACOLOGY OF α-2 AGONISTS

HISTORY

Since the early 1970s, α2-adrenergic receptor agonists have been used successfully to treat patients with hypertension and patients withdrawing from long-term abuse of drugs or alcohol. α2 -agonists produce diverse responses including analgesia, anxiolysis, sedation and sympatholysis, each of which has been reported in the treatment of surgical and chronic pain patients.

Recently the Food and Drug Administration registered two novels a2-adrenergic agonists. A role has been found for epidural clonidine in the management of pain in a variety of clinical settings. Dexmedetomidine has been registered for

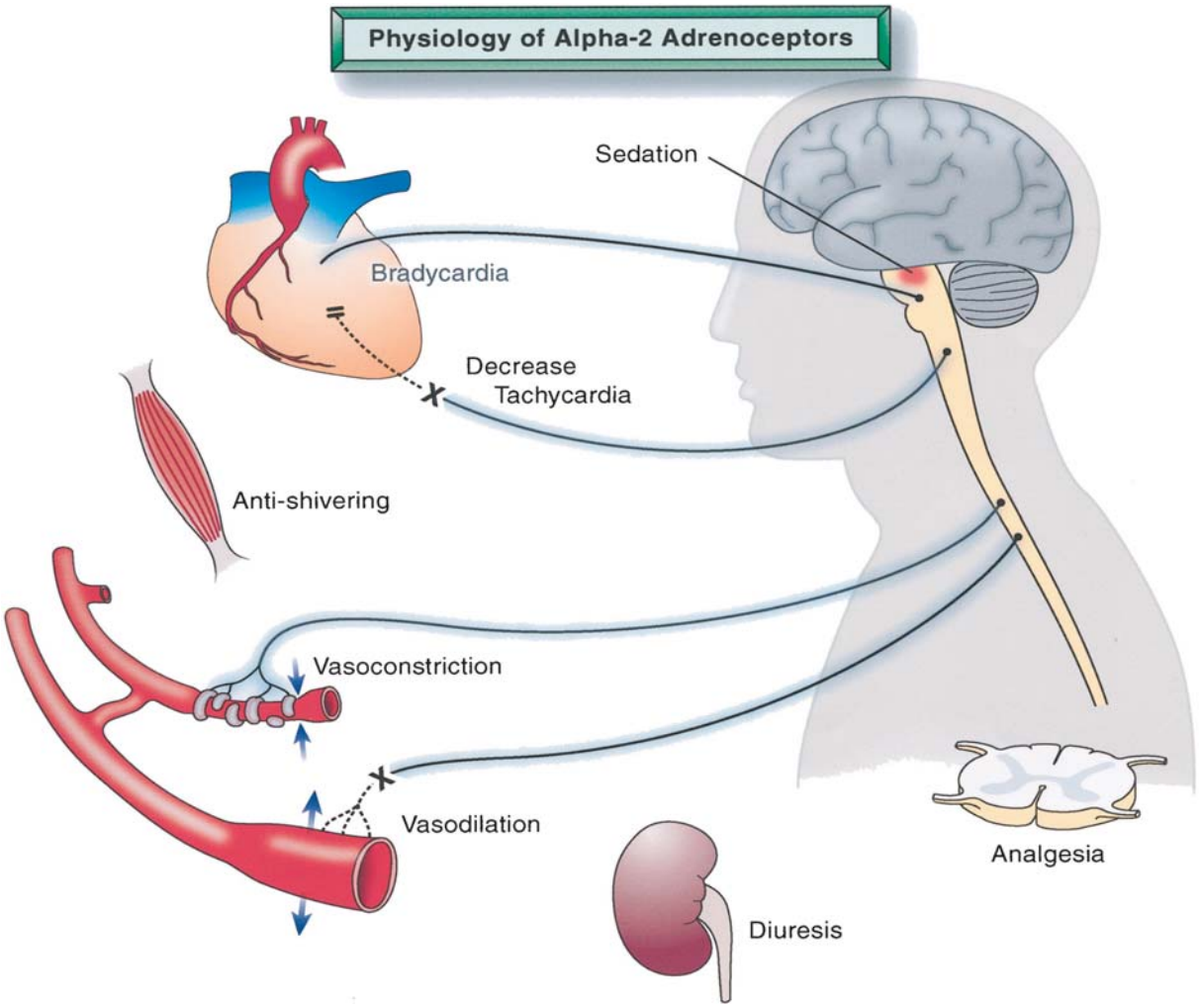
use as a sedative–analgesic in the intensive care setting. In addition to this approved setting, α2 agonists are now being studied extensively in several other perioperative settings.

α 2 receptors :

α 2a - Gene found on chromosome 10 - predominant subtype involved in sedation and analgesia.

α 2b - Gene found on chromosome 4 - predominant subtype involved in the hemodynamic effects.

α 2c - Gene found on chromosome 2



1. Activation of inhibitory G proteins causing a decreasing in cAMP.
2. Activation of G proteins that act directly on membrane bound ion channels, especially potassium channels.
3. Activation of the Nitric Oxide, cGMP pathway to cause inhibition of noradrenaline release within neuronal tissue.

a. In the medullary dorsal motor complex causing hypertension and bradycardia.

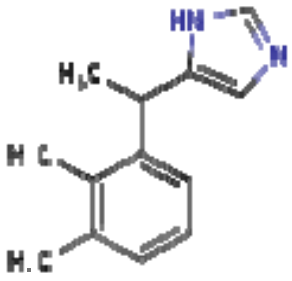
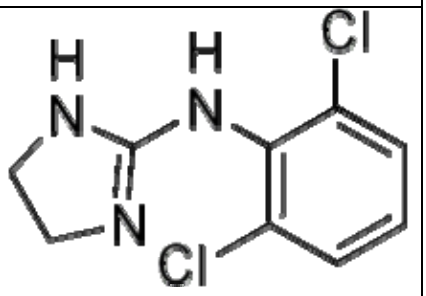
b. In the locus coeruleus leading to sedation and analgesia.

c. A high density exists in the vagus nerve, intermediolateral column and the substantia gelatinosa.

d. The dorsal horn of the spinal cord.

e. Primary sensory neurons.

Comparative pharmacology between Dexmedetomidine and Clonidine:

	Dexmedetomidine	**Clonidine**
Pharmacological profile	Imidazole derivative. Active s-enantiomer of meditomidine.	Imidazoline derivative. Partial agonist with high affinity and high intrinsic activity at α2 receptors, especially α2A receptors in the brainstem.
Structure		
α2:α1	1600:1	200:1

selectivity		
Pharmacokinetics		
Distribution	95% protein bound 1.5 l/kg volume of distribution	20% protein bound 1.5-2 l/kg volume of distribution
T1/2α	6 minutes	11 minutes
T1/2β	2-3 hours	8-12 hours
Metabolism	Conjugation (41%),N-methylation(21%).Inactive metabolites excreted in urine and faeces	½-1/3 of oral dose is excreted unchanged in urine and rest as metabolites
Pharmacodynamics		
Central nervous system		
Sedation	Unique sedative quality-patient will be sedated but arousable without respiratory depression	

Cardiovascular	Reduces HR and SVR. There is a biphasic response on blood pressure.	On rapid i.v injection-BP raises transiently due to activation of postsynaptic peripheral activation of vasoconstrictor α2A receptors.
Respiratory	Reduces minute ventilation, retain hypercapnic ventilator response	Minimal depressant effects on ventilation
Routes of administration	Intravenous,intramuscular,epidural,intrathecal,intranasal	Oral,intravenous,intramuscular,epidural,intrathecal
Dosage	Loading dose-0.5-1.0µg/kg over 10 minutes. Maintenance dose-0.3-0.7µg/kg/hr	Narrow therapeutic window phenomenon- 0.2-2.0ng/ml 4 mg/kg to a maximum of 600 mg intravenous or oral dose

		0.3 mg/kg/hr infusion Epidural 1 mg/kg
Uses	1.ICU sedation in mechanically ventilated patients 2.In anaesthesia a. before induction-at a dose of 0.3-0.67µg/kg given before 10-15 minutes ,attenuates the hemodynamic response to intubation b.as a premedication 2.5µg/kg c. sedation during regional anaesthesia	1.potent antihypertensive 2.Atention deficit hyperkinetic disorder 3.opioid withdrawal and alcohol withdrawal syndrome 4.to control loose motion due to diabetic neuropathy 5.clonidine suppression test for phaeochromocytoma

	d. as an adjuvant in bariatric surgery, craniotomy aneurysm, sleep apnea patient. E.Used for securing the airway during fibreoptic intubation.	

Alpha 2 antagonist:

Atipamezole:

Scheinin *et al.* reported about the ability of atipamezole, a novel selective a2-adrenoceptor antagonist, to reverse the sedative properties of dexmedetomidine in volunteers. Both the sedative and the sympatholytic effects of intramuscular dexmedetomidine were dose-dependently antagonized by intravenous atipamezole. However, the sensitivity for reversal of these two responses may be different. Because the agonist and the antagonist have similar elimination half-lives, the likelihood of recurrence of the clinical effects of dexmedetomidine after reversal by atipamezole is small. Therefore the a2 agonists provide a titratable form of hypnotic sedation that can be reversed readily.

REVIEW OF LITERATURE

1. Sukhminder Jit Singh Bajwa et.al [42] conducted a study to compare two α-2 adrenergic agonists dexmedetomidine and clonidine in epidural anaesthesia. A prospective randomized trial was carried out which included 50 adult female patients between 44-65 years of age, ASA I/II grade who underwent vaginal hysterectomies. The patients were randomly allocated into two groups; ropivacaine + dexmedetomidine (RD) and ropivacaine + clonidine (RC), comprising of 25 patients each. Group RD received 17 ml of 0.75% epidural ropivacaine and 1.5µg/kg of dexmedetomidine and group RC received 17 ml of ropivacaine and 2µg/kg of clonidine. Onset of analgesia, sensory and motor block levels, sedation, duration of analgesia and side effects were observed. Statistically the demographic profile, initial and post operative block characteristics and vital signs were comparable and non-significant in both groups. Sedation scores were better with dexmedetomidine than clonidine and statistically significant ($P < 0.05$). The side effect profile was also comparable with a little higher incidence of nausea and dry mouth in both the groups which was statistically non significant ($P > 0.05$). To conclude dexmedetomidine is a better neuraxial adjuvant than clonidine for providing early onset of sensory analgesia, adequate analgesia and a prolonged post operative analgesia.

2.**Ashraf Abdul Baki Abdul Baset .M.D**[40] conducted a study to evaluate the analgesic properties of dexmedetomidine alone or in combination with bupivacaine 0.125% when administered as an epidural infusion for the treatment of postoperative pain in patients undergoing extensive abdominal surgery. Ninety adult patients scheduled for abdominal surgery were studied. An epidural catheter was inserted in all patients at the T8-T9 vertebral interspace and a 15 ml of 0.5% bupivacaine injected epidurally. General anaesthesia was induced with propofol and atracurium and was maintained with a sevoflurane. VAS was used for assessment of postoperative pain and when it reaches 30, patients were then randomly divided into three groups to receive an epidural infusion at 10 mL/h of bupivacaine 0.125%, dexmedetomidine 0.5 μg/kg/hr in bupivacaine 0.125% and dexmedetomidine 0.5 μg/kg/hr diluted in 0.9% saline in Group B, Group DB, and Group D, respectively. VAS score, sedation score, sensory and motor blockade, MAP and HR were monitored and recorded. Rescue analgesia was given in the form of patient controlled analgesia and the total morphine requirements were recorded together with the time to first analgesic requirement. The Group DB having the lowest morphine requirements (11.9 ± 15.6 mg) and the longest interval before analgesia was requested (12.0 ± 6.5 hr) while Group B, had the shortest period (4.0 ± 4.6) and the highest requirement (34.9 ± 20.7) $P < 0.05$. VAS were generally satisfactory for all groups, the mean score being below 30 mm. MAP and HR were similar in

groups (D and DB), both were significantly lower than in Group B (P< 0.05), however such difference was not thought to be of clinical importance. The motor block was more significant in-group B and DB compared with group D in the PACU and up to 6 h post infusion (P <0.05 compared with dexmedetomidine group) While sensory blockade was more pronounced in the combination group. He concluded that patients undergoing abdominal surgery, the addition of the α_2-adrenergic agonist dexmedetomidine to epidural infusions of bupivacaine significantly improved postoperative analgesia without significant side effects.

3. Antonio Mauro Vieira et.al[38] conducted a study to evaluate the analgesia and sedation promoted by clonidine or dexmedetomidine associated to epidural ropivacaine in the postoperative period of subcostal cholecystectomy. Clonidine and dexmedetomidine are α-2-adrenergic agonists with analgesic proprieties which potentiate local anaesthetic effects when epidurally administered. Forty patients of both gender were participated in this randomized double-blind study, aged 18 to 50 years, weighing 50 to 100 kg, physical status ASA I or II, submitted to subcostal cholecystectomy. The subjects were distributed in two groups: Clonidine (CG) received clonidine (1 mL = 150 μg) associated to 0.75% epidural ropivacaine (20 mL); Dexmedetomidine (DG) received dexmedetomidine (2 μg/kg) associated to 0.75% epidural ropivacaine (20 mL). Analgesia and sedation were evaluated 2, 6 and 24 hours after anesthetic

recovery. Both groups presented some grade of sedation at 2 and 6 hours, with statistically significant difference between the two moments for the dexmedetomidine group. There has been analgesia in both groups, especially at 2 and 6hours. There has been statistically significant difference among periods of 2, 6 and 24 hours in the dexmedetomidine group; in the clonidine group, this statistically significant difference was observed between the periods of 2 and 6 hours and between 2 and 24 hours. To conclude that the association of clonidine or dexmedetomidine to 0.75% ropivacaine induces analgesia and sedation in 2 and 6 hours after anaesthetic recovery in patients submitted to subcostal cholecystectomy and that clonidine promotes more prolonged analgesia.

4. Taylor Brandao Schnaider., et.al [37] studied to evaluate the effects of epidural ketamine, clonidine and dexmedetomidine, in patients undergoing upper abdominal surgery. Low dose ketamine decreases nociception by blocking NMDA receptor channels. Alpha2- adrenergic receptor activation triggers intense analgesic response .A randomized double-blind study 70 patients of both genders, aged 18 to 50 years, physical status ASA I or II, submitted to subcostal cholecystectomy under general anaesthesia associated with lumbar epidural anaesthesia. Lumbar epidural anaesthesia was randomly induced as follows: Control group: 20 mL of 0.75% ropivacaine and 1 mL of 0.9% saline solution (n = 10); Ketamine group: 20 mL of 0.75% ropivacaine

and 0.5 mg/kg ketamine (n = 20); Clonidine group: 20 mL of 0.75% ropivacaine and 1 mL clonidine (150 μg) (n = 20); Dexmedetomidine group: 20 mL of 0.75% ropivacaine and 2 μg/kg dexmedetomidine (n = 20). Anaesthesia was induced with etomidate, alfentanil and rocuronium and was maintained with isoflurane and alfentanil. Analgesia was evaluated by clinical signs and inhalational anaesthetic inspired concentration was evaluated by anaesthetic gases analysis during surgery. All patients receiving ketamine, clonidine or dexmedetomidine had heart rate and systemic blood pressure decrease and have not required perioperative analgesic complementation. For the same patients, isoflurane inspired concentration varied from 0.5 vol% to 1vol% and there were no clinical signs or responses suggesting inadequate anaesthetic levels. To conclude epidural ketamine, clonidine or dexmedetomidine decreases alfentanil consumption and isoflurane inspired concentration in the intraoperative period of upper abdominal surgery.

5**. Vijay.G.Anand et.al**[41] conducted a study to compare the effects of caudal dexmedetomidine combined with ropivacaine to provide post operative analgesia in children. The study was conducted in 60 children who had undergone lower abdominal surgeries. They were allocated into 2 groups of 30 each. Group RD received 0.25% ropivacaine 1 ml/kg with dexmedetomidine 2μg/kg (made up to 0.5ml) and group R received 0.25% ropivacaine 1ml/kg +

0.5 ml normal saline. Induction was done with 50%$N_2O$and 8% sevoflurane in O_2 in spontaneous ventilation and then LMA was inserted .After that caudal block was performed and the study drug was given as mentioned above. The duration of post operative analgesia was recorded and median of 5.5 hrs in Group R compared with 14.5 hours in Group RD, with a p value of <0.001.Group R patients achieved and statistically significant higher FLACC score compared to RD patients. The mean sedation score, emergence behaviour score, mean emergence time was statistically highly significant (<0.001).The peri operative hemodynamics were stable in both groups. To conclude caudal dexmedetomidine (2µg/kg) with 0.25% ropivacaine 2ml/kg for paediatric lower abdominal surgeries achieved significant post operative pain relief that resulted in a better quality of sleep and prolonged duration of arousable sedation.

6. A.M.El-Hennawy., et.al [12] studied to compare the analgesic effects and side-effects of dexmedetomidine and clonidine added to bupivacaine in paediatric patients undergoing lower abdominal surgeries. Sixty patients (6 months to 6 yr) were evenly and randomly assigned into three groups in a double-blinded manner. After sevoflurane in oxygen anaesthesia, each patient received a single caudal dose of bupivacaine 0.25% (1 ml/kg) combined with either dexmedetomidine 2 µg/kg in normal saline 1 ml, clonidine 2 µg/ kg in normal saline 1 ml, or corresponding volume of normal saline according to

group assignment. Hemodynamic variables, end-tidal sevoflurane, and emergence time were monitored. Postoperative analgesia, use of analgesics, and side-effects were assessed during the first 24 h. Addition of dexmedetomidine or clonidine to caudal bupivacaine significantly promoted analgesia time [median (95% confidence interval, CI): 16 (14–18) and 12 (3–21) h, respectively] than the use of bupivacaine alone [median (95% CI): 5 (4–6) h] with $P<0.001$. However, there was no statistically significant difference between dexmedetomidine and clonidine as regards the analgesia time ($P=0.796$). No significant difference was observed in incidence of hemodynamic changes or side-effects. To conclude the addition of dexmedetomidine or clonidine to caudal bupivacaine significantly promoted analgesia in children undergoing lower abdominal surgeries with no significant advantage of dexmedetomidine over clonidine and without an increase in incidence of side-effects.

7. Mausumi Neogi et.al[39] conducted a randomized prospective study was designed to assess and compare the efficacy of clonidine and dexmedetomidine used as adjuvant to ropivacaine for caudal analgesia in paediatric patients. Seventy five patients undergoing elective inguinal herniotomy were included in one of the three following groups. Group R patients received 1 ml /kg of 0.25% ropivacaine caudally. Group C patients received 1ml kg-1 of 0.25% ropivacaine

and 1ml / kg clonidine. Patients of group D were given 1 ml/ kg of 0.25% ropivacaine and 1 μg.kg-1 dexmedetomidine. Postoperative analgesia was assessed by CRIES scale.

The mean duration of analgesia was 6.32±0.46 hours in group R, 13.17±0.68 hours in group C and 15.26±0.86 hours in group D. The prolongation of duration of analgesia was significant in both groups C and D in comparison to group R. The incidences of adverse effects were statistically insignificant between the three groups.

They concluded that addition of both clonidine and dexmedetomidine with ropivacaine administered caudally significantly increase the duration of analgesia.

8.Mamta sethi et.al[43] studied to evaluate the effect of adding small dose ketamine in a multi modal regimen of post operative patient controlled epidural analgesia in 100 patients who had undergone major upper abdominal surgery belonging to ASA I/II. They were randomly allocated into two groups. After extubation, the patient was shifted to Post Anaesthesia Care Unit. In PACU, the patient was assessed for pain intensity using the 10 point Visual Analogue Scale. If VAS ≥ 3, an initial dose of 0.125% bupivacaine 10 ml was administered via epidural catheter. A PCEA pump was then attached. Group I received bupivacaine 0.0625% and morphine sulphate (preservative free)

0.05mg/ml. Group II received bupivacaine 0.0625%, morphine sulphate (preservative free) 0.05mg/ml and ketamine hydrochloride (preservative free) 0.2mg/ml. The mean morphine consumption in group I after first and second post operative day was 8.38± 2.85 and 7.64±1.95 mg respectively compared to 6.81±1.35 and 6.25±1.22 mg ($P<0.05$) in group II. Pain relief at rest and at movement after 6, 12, 24 and 48 hours post operatively was significantly better in group II ($P<0.05$) than in group I.

9. Jum Carren, M.D., et.al studied the cardiovascular effects, sedation and quality of analgesia between two different drug mixtures for epidural anaesthesia. A double blinded randomized clinical trial including 40 ASA I, II adult patients who were scheduled for open elective abdominal surgery under epidural anaesthesia. There were two groups: 1) Dexmedetomidine 1μg/kg plus 2% Lidocaine with epinephrine 5 mg/kg and 2) Fentanyl 100 μg/kg plus 2% Lidocaine with epinephrine. The main outcomes measured were: heart rate, systolic arterial pressure, diastolic arterial pressure, mean arterial pressure, peripheral oxygen saturation, respiratory rate, VAS score, highest dermatome blocked, Bromage score and Ramsay score every 5 minutes the first 15 minutes after the initial epidural dose and every 10 minutes until the end of surgery. They also evaluated the incidence of shivering, nausea, vomiting and inadequate anaesthesia as well as the need of Atropine and second dose of local anaesthetic. There were no differences in demographic characteristics between groups. They

found higher Ramsay scores in the Dexmedetomidine group after the first 20 minutes of the epidural injection, with a mean score of 3.7 vs. 2.7 (P= 0.001), 30 minutes (3.8 vs. 2.9, P=0.002), 40 minutes (4.25 vs. 2.9, P= 0.001), 50 minutes (4.25 vs. 2.7, P= 0.001), 60 minutes (3.84 vs. 2.71, P= 0.004), 70 minutes (3.76 vs. 2.84, P= 0.02) and 80 minutes (3.42 vs. 2.46, P=0.001). There was also a lower pulse rate in this group during the same period of time. The mean heart rate at 20 minutes was lower in Dexmedetomidine group with 73.45 beats/min vs. 86.05 beats/min in Fentanyl group (P= 0.016). They found the same differences at 50 minutes (70.75 vs. 82.05, P= 0.041) and 60 minutes (71.52 vs. 85.85, P= 0.018). They did not find significant differences in systolic, diastolic or mean arterial pressure, as well as in peripheral oxygen saturation, respiratory rate, height of sensitive blockade or motor level. They have concluded that when administered in the epidural space for abdominal surgery, Dexmedetomidine does not increase the number of dermatomes blocked or motor blockade. It only produces deeper sedation, with the concomitant decrease in pulse rate that needed treatment with atropine.

AIM OF THE STUDY

To compare the efficacy and safety of two α-2 adrenergic agonists, Dexmedetomidine and Clonidine in post-operative epidural analgesia after upper abdominal surgery with respect to their sedative properties and hemodynamic stability.

MATERIALS AND METHODS

This study was conducted at Rajiv Gandhi Government General Hospital, Chennai-600 003 between August 2011-October 2011 after obtaining approval from hospital ethical committee.

Study design: A prospective single- blinded, randomized, controlled study.

Sample size: 50 patients were studied.

Inclusion criteria: ASA I & II patients aged 18-60 years undergoing upper abdominal surgery under general anaesthesia and planned for an epidural analgesia post operatively.

Randomisation: Patients were randomly allocated into 2 groups of twenty five each Group C (Clonidine) and Group D (Dexmedetomidine). Randomisation was done by lottery method.

EXCLUSION CRITERIA:

Patients with coagulation abnormalities

Patients with cardiac or renal disease

Patients with neurological illness

Patients with mental illness

Patients with deformity of spinal column

Patients with allergy to local anaesthetics

Patients not fitting into inclusion criteria

Pre-operative visit:

In all patients, age, body weight and baseline vital parameters were recorded. History regarding previous anaesthesia, surgery and significant other co morbid illness, medications and allergy was recorded. Complete physical examination and airway assessment were done.

In the preoperative period all patients were instructed about the benefits of epidural analgesia and 10-point visual analogue scale. And also informed consent form was obtained from all the study group patients.

Premedication:

All patients were premedicated with T.Alprazolam 0.25-0.5mg, T.Perinorm 10mg and T.Ranitidine 150 mg at 6 am on the day of surgery.

Monitors attachment and intravenous access:

Standard monitors like ECG, Non-invasive BP, and spO2 were connected to the patient. Intravenous access was done using 16 or 18 Gauge venflon and intravenous crystalloid was started.

Epidural Catheterisation:

Prior to induction of anaesthesia under strict aseptic precaution an epidural catheter tip was kept around T6-T8 intervertebral space in lateral position by using 9 cm long, 17 gauge Tuohy's needle by loss of resistance technique. The epidural catheter position was rechecked with a test dose containing 3 ml of 1.5% lignocaine with adrenaline (5µg/ml).

General Anaesthesia:

In all patients routine general anaesthesia with controlled ventilation was used. Anaesthesia was induced with thiopentone sodium 5mg/kg and fentanyl citrate 2µg/kg and tracheal intubation was done with atracurium besylate 0.6mg/kg body weight.

Anaesthesia was maintained with atracurium besylate and sevoflurane 1-2 % in N2O –O2 mixture to maintain muscle relaxation and depth of anaesthesia respectively. Intra operative analgesia was achieved with intermittent boluses of fentanyl citrate. After the end of procedure, patient was reversed with neostigmine and glycopyrrolate.

Study Drug Administration:

Just before extubation, the study drug was given via epidural catheter after the negative aspiration for CSF and blood.

Group C: This group received 7ml of 0.125% bupivacaine + 2µg/kg clonidine made upto 3 ml with normal saline.

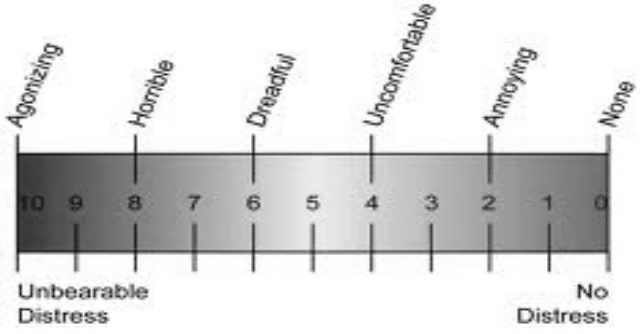
GroupD: This group received 7 ml of 0.125% bupivacaine + 2 µg/kg of dexmedetomidine made upto 3 ml with normal saline.

Patients were observed in the theatre for 15 minutes and then shifted to Post Anaesthesia Care Unit (PACU) for monitoring.

Monitoring in Post Anaesthesia Care Unit:

Various parameters like HR, Blood pressure (both systolic and diastolic), SPo2, Visual Analogue Scale (VAS) and Ramsay Sedation Score (RSS) were observed for 24 hours post operatively. Incidences of side effects were also noted. Injection Tramadol 50 mg IV was used as rescue analgesia when pain score was more than 4 (i.e. VAS ≥4)

ASSESSMENT OF PAIN USING VISUAL ANALOGUE SCORE



ASSESSMENT OF SEDATION USING MODIFIED RAMSAY SCORE

Awake levels

1. Anxious, agitated or both
2. Co-operative oriented, tranquil
3. Response to commands only

Asleep levels

4. Brisk response to loud auditory stimulus
5. Sluggish response to loud auditory stimulus
6. No response to loud auditory stimulus

Vitals monitoring:

Vital parameters were monitored continuously and recordings were made every 5 minutes until 1 hour and at every 15 minutes interval for next hour and finally at 60 minutes interval for next 22 hours. Hypotension (defined as systolic arterial pressure falling less than 90mmHg) was treated with inj.Ephedrine 6mg and bradycardia (heart rate <50 beats/min) was treated with 0.3mg of inj.Atropine.

Recording of adverse effects:

During the first 24 hours of postoperative period, adverse events like nausea, vomiting, dizziness, pruritus and respiratory depression were noted. Nausea and vomiting were treated with 4mg of intravenous ondansetron.

OBSERVATION AND RESULTS

STATISTICAL ANALYSIS

Data were analysed using INSTAT 3 (Graph Pad Software, California, USA).Two sided independent student' s *t* tests to analyse continuous data, Fisher's exact test and chi-square test for categorical data were used. $P<0.05$ was considered as statistically significant.

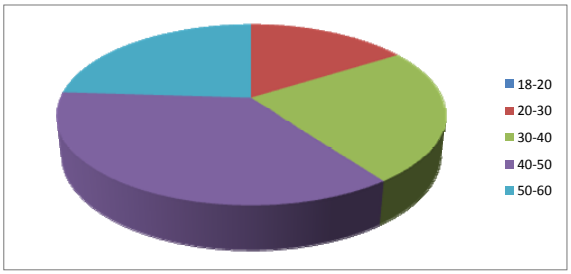
DEMOGRAPHIC DATA:

The two groups were comparable with respect to their age, weight, sex and ASA Physical status. There was no statistically significant difference among two groups in demographic profile.

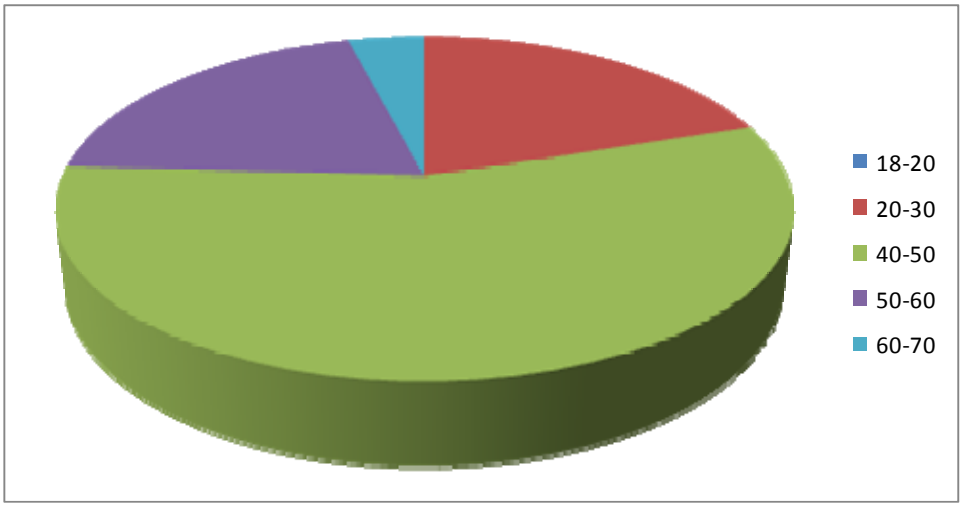
AGE (student' s t test)

	No. of cases	Mean±S.D	p value
Group D	25	42.32 ± 11.657	0.3758
Group C	25	58.24 ± 8.313	

AGE (GROUP D)



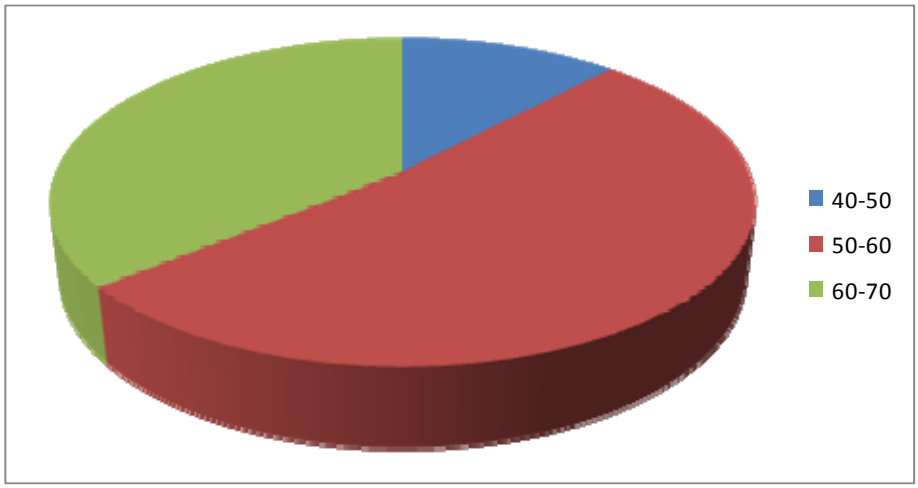
AGE (GROUP C)



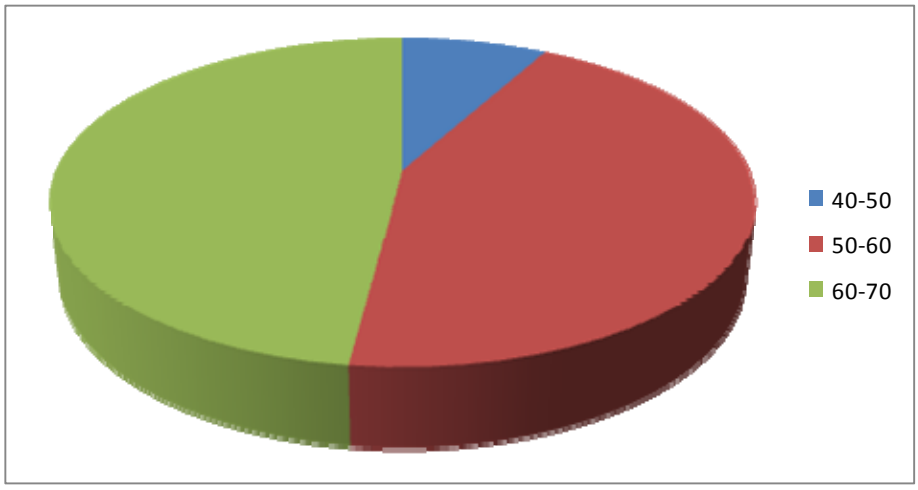
WEIGHT (student's t test)

	No. of cases	Mean±S.D	p value
Group D	25	58.24 ± 6.514	0.0001
Group C	25	59.2 ± 6.298	

WEIGHT (GROUP D)



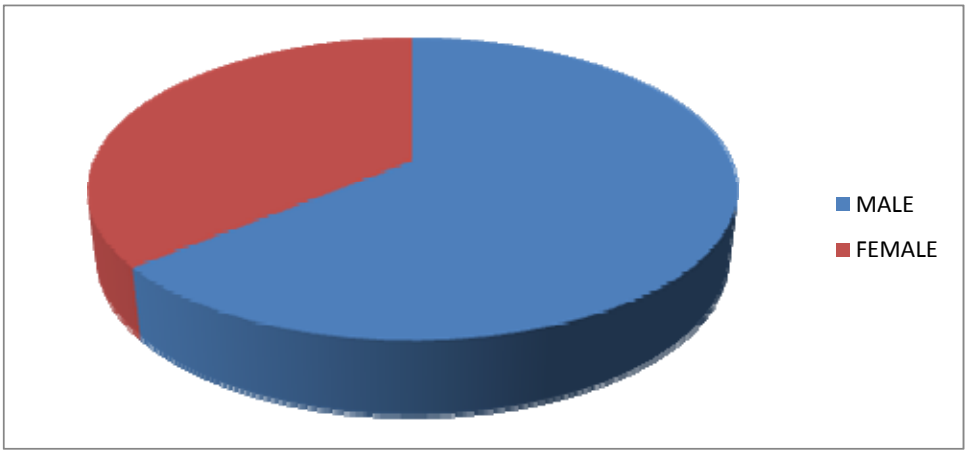
WEIGHT (GROUP C)



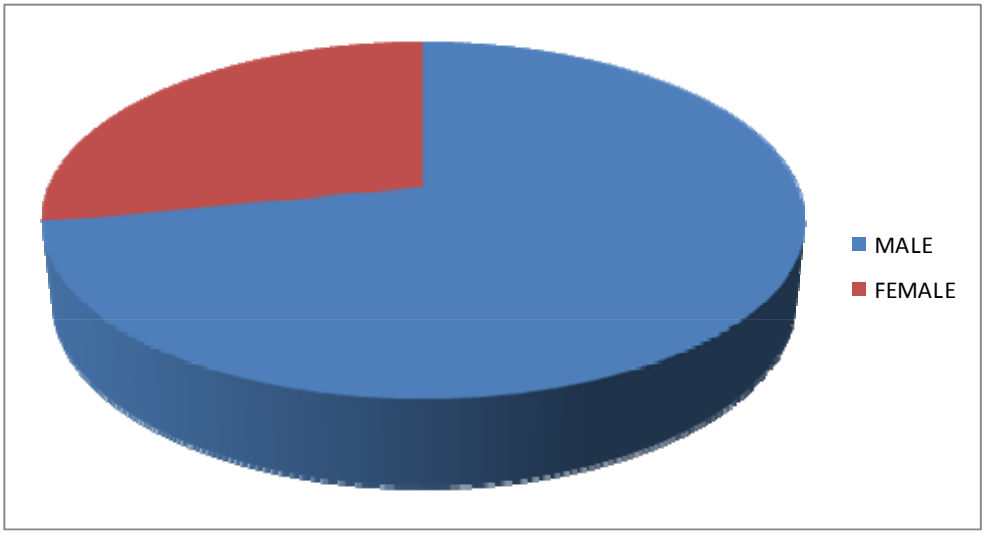
SEX (Chi-square test)

	MALE	FEMALE	p value
Group D	16(64%)	9(36%)	0.7157
Group C	18(72%)	7(28%)	

SEX (GROUP D)

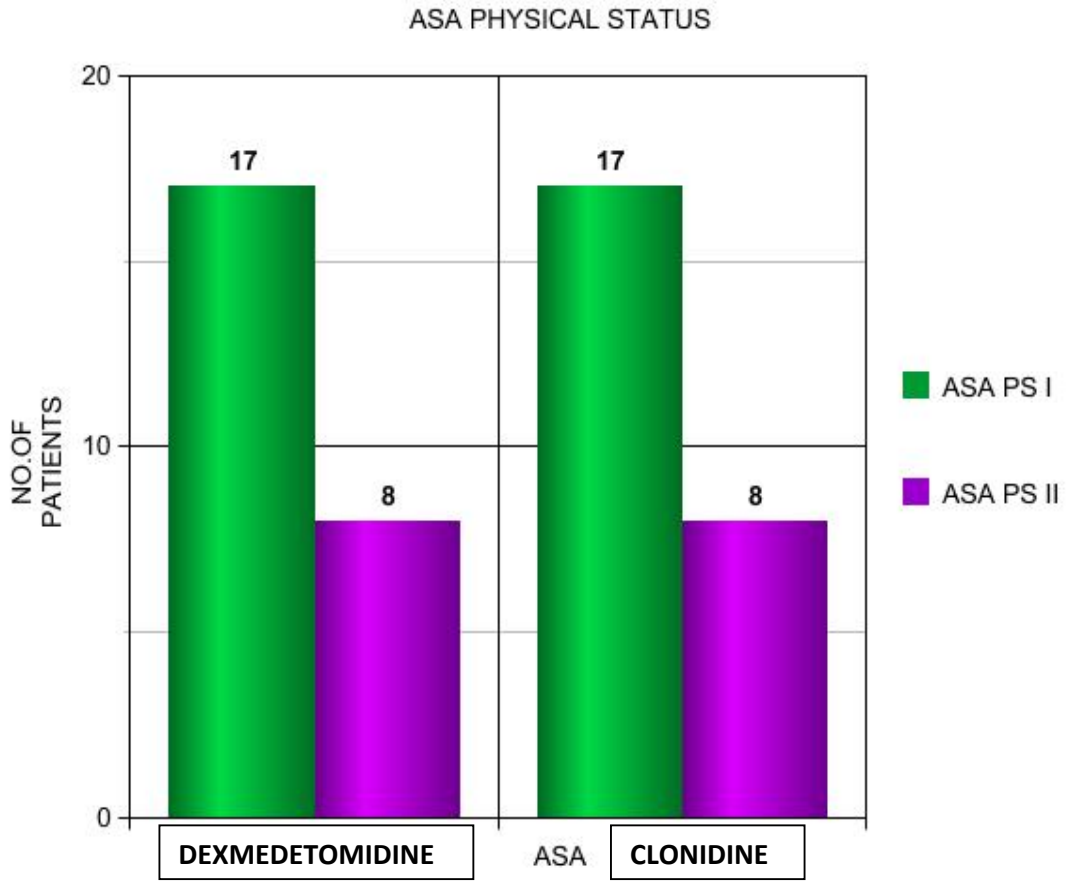


SEX (GROUP C)



ASA-PHYSICAL STATUS (Chi-square test)

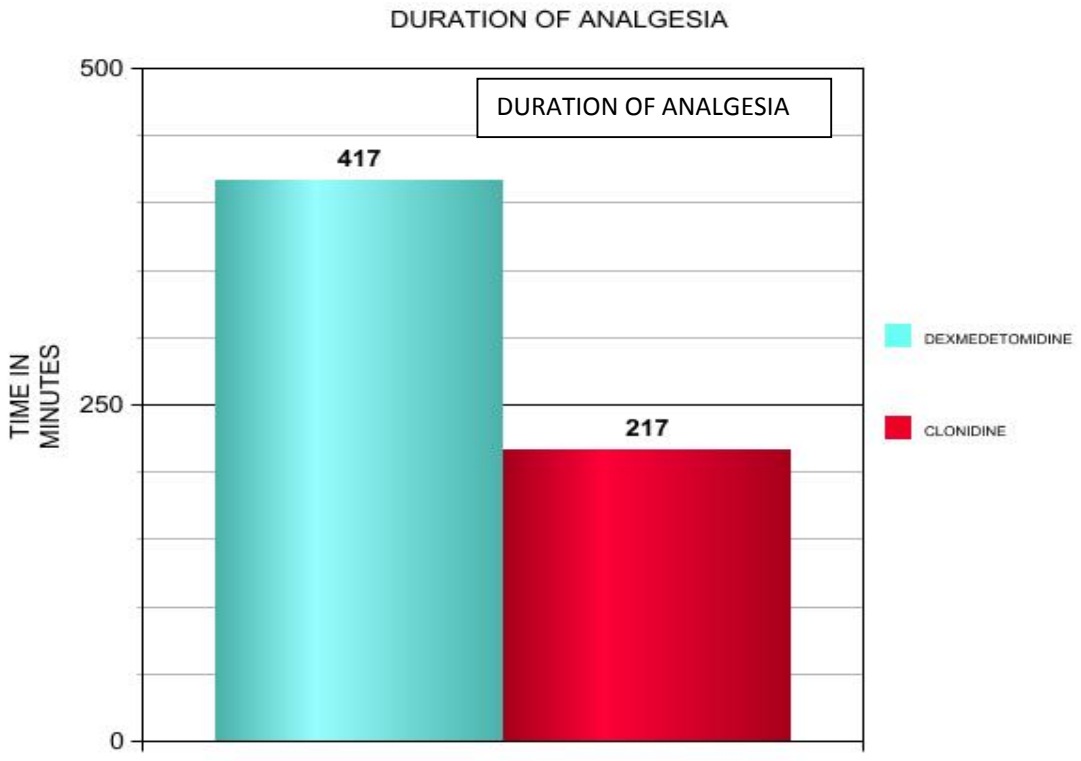
	ASA-PS I	ASA- PS II	p value
Group D	17 (68%)	8(32%)	1.000
Group C	17 (68%)	8 (32%)	



DURATION OF ANALGESIA (student's *t* test)

	No. of Cases	MEAN±S.D	p value
GROUP D	25	417.32 ± 67.36	0.0001
GROUP C	25	217.2 ± 25.82	

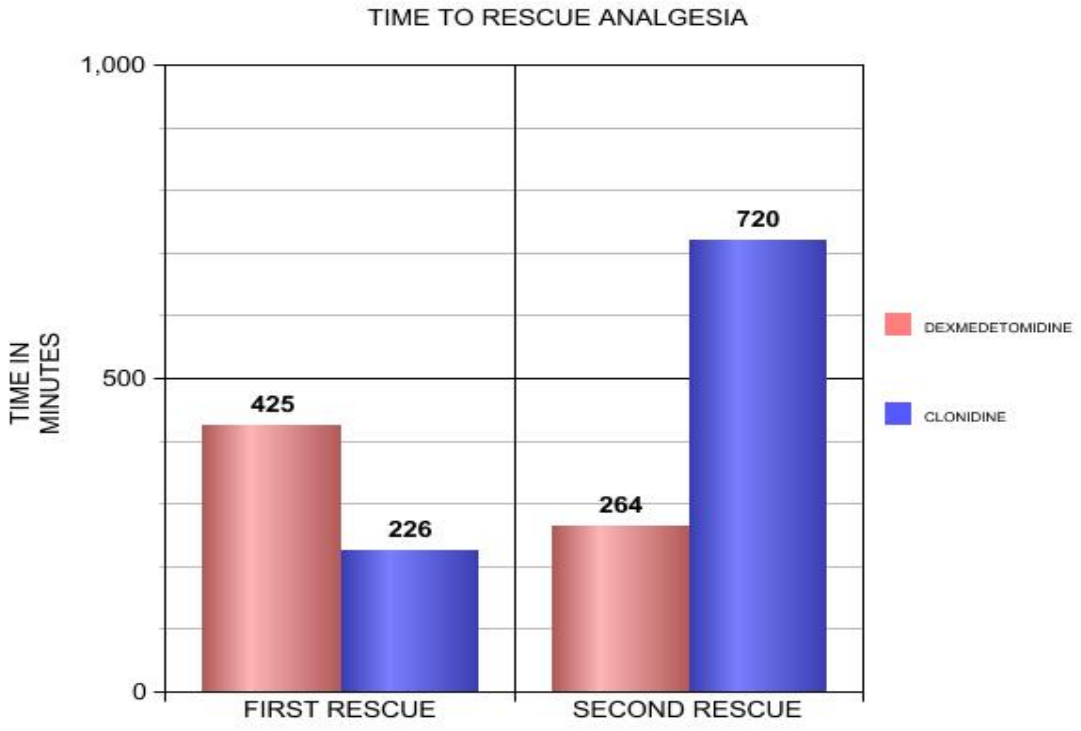
The mean duration of analgesia was 417.32±67.36 minutes in Group D and 217.2±25.32 minutes in Group C. There was statistically significant difference among two groups in the mean duration of analgesia (P<0.05).



TIME TO 1st RESCUE ANALGESIA (student's *t* test)

	No. of Cases	MEAN±S.D	p value
GROUP D	25	425.6 ± 64.27	0.0001
GROUP C	25	226 ± 24.83	

The mean time for 1st rescue analgesia (defined as the time at which patient demands some mode of pain relief i.e. when VAS score more than 4) was 425.6±64.27 minutes in Group D and 226±24.83 minutes in Group C. There was significant difference among two groups in the duration of time for rescue analgesia (P<0.05).



TIME TO 2ND RESCUE ANALGESIA (student's *t* test)

	No. of Cases	MEAN±S.D	p value
GROUP D	25	264.8 ± 396.25	0.0001
GROUP C	25	720.4 ± 226.83	

The mean time for 2nd Rescue analgesia (defined as the time at which the patient demands some mode of pain relief from the time of 1st rescue analgesia (i.e VAS becomes more than 4) was 264.8±396.25 minutes in Group D and 720.4±226.83 minutes in Group C. There was significant difference among two groups in the duration of time for rescue analgesia ($P<0.05$).

VISUAL ANALOGUE SCORE (student's t test)

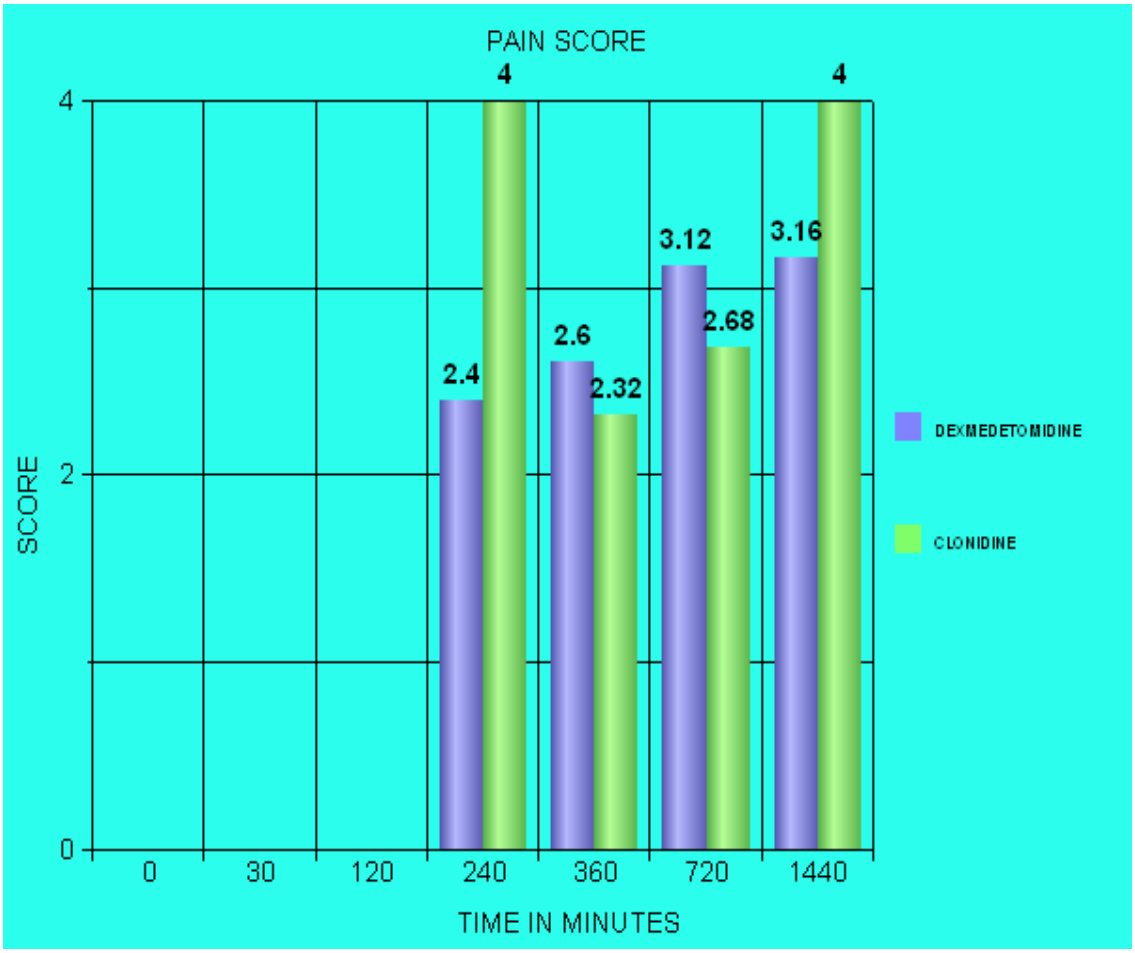
Time in Minutes	No. of Cases	GROUP D Mean ± SD	GROUP C Mean ± SD	p value
0	25	0.44 ± 0.65	0.48 ± 0.58	0.820
30	25	0.08 ± 0.27	0.12 ± 0.33	0.644
120	25	0.24 ± 0.59	0.28 ± 0.54	0.805
360	25	0.84 ± 0.89	1.76 ± 0.99	0.001
720	25	2.96 ± 1.01	2.08 ± 1.07	0.004
1440	25	3.48 ± 0.82	3.52 ± 1.04	0.881

There was no difference in pain score at 0,30 and 120 minutes and was found to be statistically not significant ($p>0.05$).

At 360 minutes, the mean VAS score in Group D was 0.84±0.89 and in Group C was 1.76±0.99; there was statistical significant difference in both groups ($p<0.05$)

The mean VAS score in Group D was 2.96±1.01 and in Group C was 2.08±1.07 at 720 minutes which was found to be statistically significant ($p<0.05$)

At 1440 minutes, the mean VAS score in Group D was 3.48±0.82 and in Group C was 3.52±1.04 and was found to be statistically not significant (p>0.05).



RAMSAY SEDATION SCORE (student's t test)

Time in Minutes	No. of Cases	GROUP D Mean±S.D	GROUP C Mean±S.D	p value
0	25	3 ± 0	3 ± 0	1.000
60	25	3 ± 0	3 ± 0	1.000
120	25	3 ± 0	2.76 ± 0.43	0.007
180	25	3 ± 0	2.76 ± 0.43	0.007
240	25	2.84 ± 0.37	2 ± 0	0.001
300	25	2.84 ± 0.37	2 ± 0	0.001
360	25	2.56 ± 0.50	2 ± 0	0.001

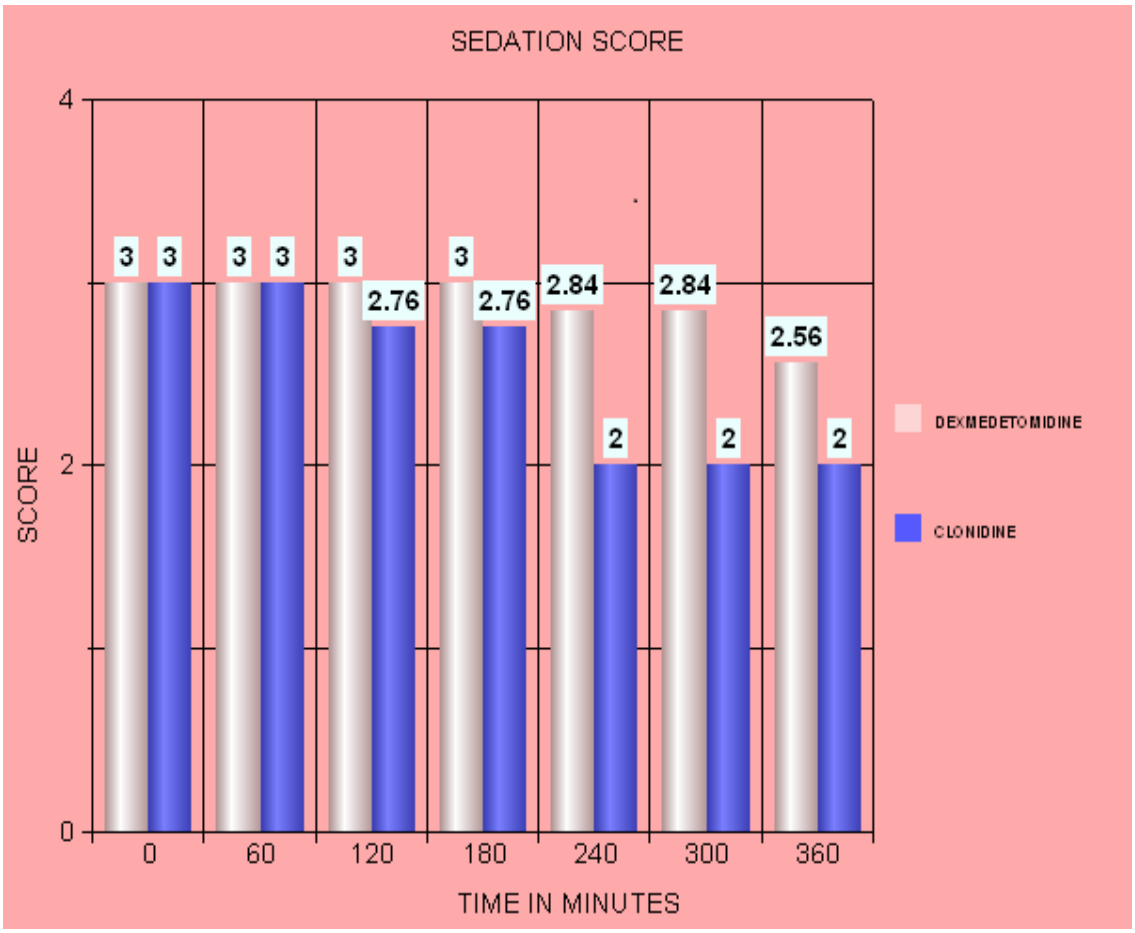
There was no statistical difference in Ramsay Sedation Score in both groups at 0 and 60 minutes ($p>0.05$).

The mean sedation score in Group D was 3±0 and in Group C was 2.76±0.43 at 120 and 180 minutes and was found to be statistically Significant ($p<0.05$).

At 240 and 300 minutes, the mean sedation score in Group D was 2. 84±0.37

and in Group C was 2±0 minutes which was found to be statistically significant ($p<0.05$)

The mean sedation score in Group D was 2.56±0.50 and in Group C was 2±0 at 360 minutes ,which was found to be statistically significant ($p<0.05$)

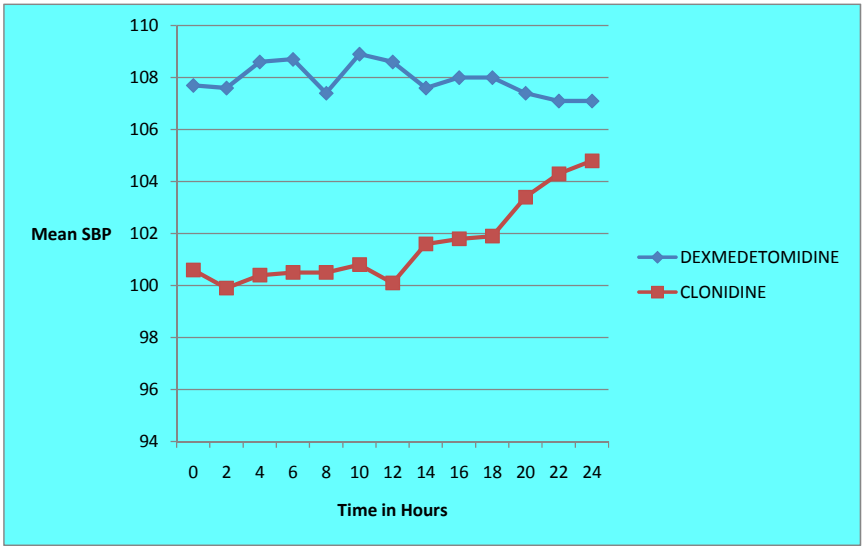


HEMODYNAMICS

SYSTOLIC BLOOD PRESSURE (student's *t* test)

	No. of Cases	MEAN±S.D	p value
GROUP D	25	107.32±7.93	
GROUP C	25	101.4±3.21	0.0012

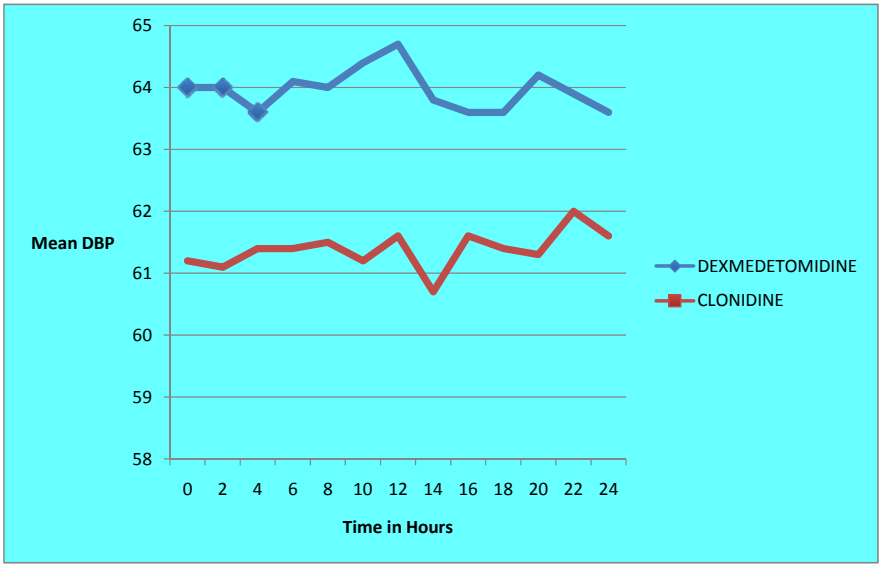
The mean systolic blood pressure in Group D was 107.32±7.93mmHg and in Group C was 101.4±3.21mmHg.There was significant statistical difference in systolic blood pressure between the two groups ($p<0.05$).



DIASTOLIC BLOOD PRESSURE (student's *t* test)

	No. of Cases	MEAN±S.D	p value
GROUP D	25	63.12 ± 4.59	0.008
GROUP C	25	59.36 ± 5.04	

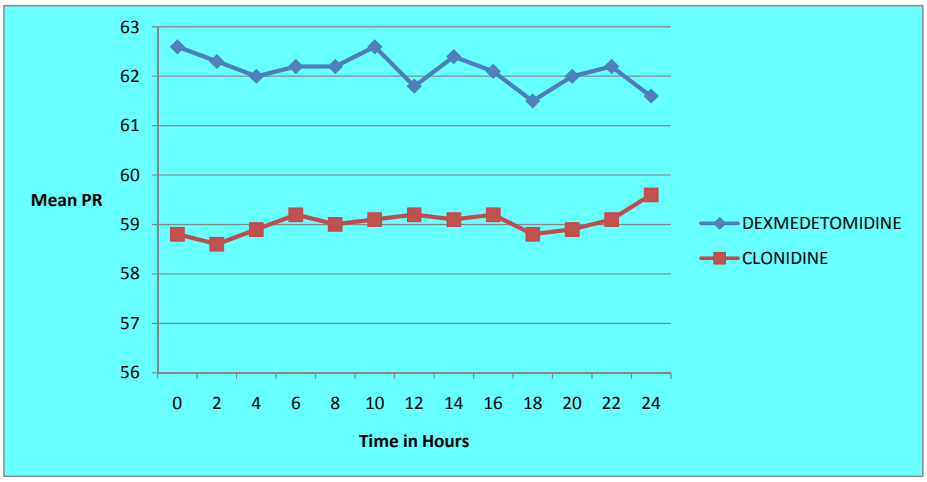
The mean diastolic blood pressure in Group D was 63.12±4.59 mm Hg and in Group C was 59.36±5.04 mmHg, which was found to be statistically significant ($p<0.05$).



HEART RATE (student's *t* test)

	No. of Cases	MEAN±S.D	p value
GROUP D	25	62.6 ± 6.04	
GROUP C	25	58.52 ± 6.04	0.028

The mean heart rate in Group D was 62.6±6.04 and in Group C was 58.52±6.04, which was found to be statistically significant ($p<0.05$).



SIDE EFFECTS (Chi-square Test)

Time in Minutes	No. of Cases	GROUP D	GROUP C	p value
Hypotension	25	2(8%)	6(24%)	0.246
Bradycardia	25	2(8%)	4(16%)	0.667
Nausea	25	0(0%)	2(8%)	0.489
Vomiting	25	0(0%)	2(8%)	0.489
Dizziness	25	2(8%)	4(16%)	0.667
Dry mouth	25	0(0%)	4(16%)	0.109
Respiratory Depression	25	0(0%)	0(0%)	1.000

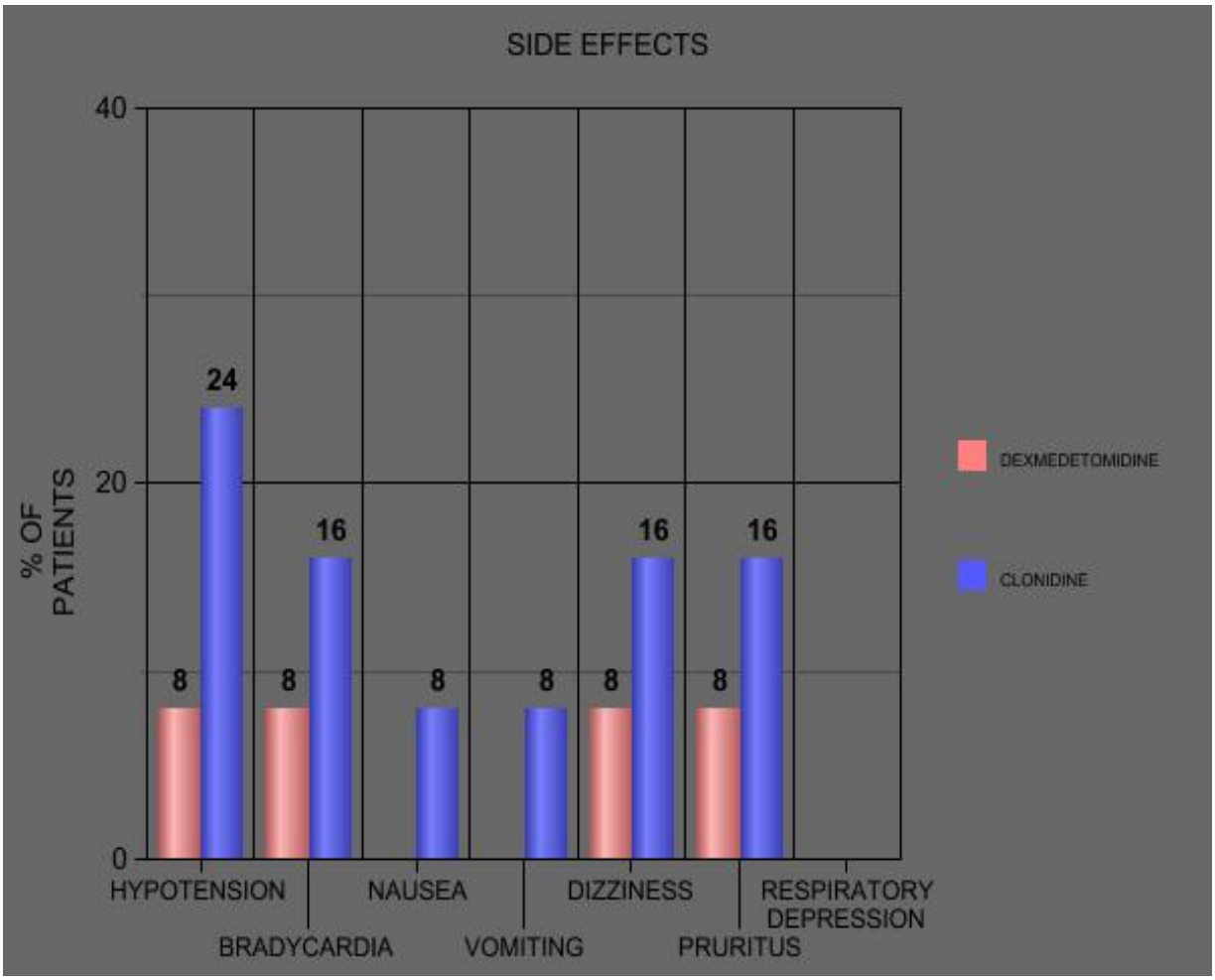
The incidence of hypotension in Group D was 8% and in Group C was 24% which was statistically not significant ($p>0.05$)

The incidence of bradycardia in Group D was 8% and in Group C was 16% and there was statistically no significant difference in both groups ($p>0.05$)

The incidence of nausea and vomiting in Group C was 8% and in Group D no patient had nausea and vomiting which was statistically not significant ($p>0.05$)

The incidence of dizziness in Group D was 8% and in Group C was 16% and there was statistically no significant difference in both groups (p>0.05)

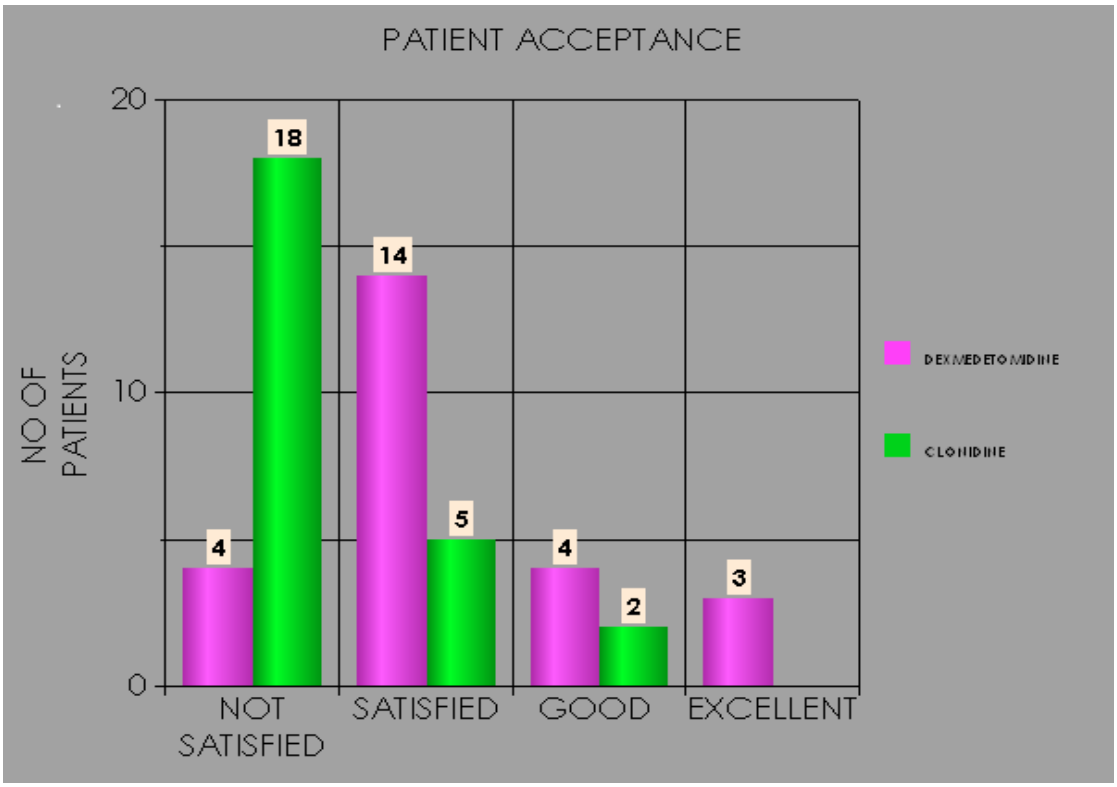
The incidence of dry mouth in Group C was 16% and in Group D, no patient had reported dry mouth. Statistically there was no significant difference in both groups (p>0.05)



PATIENT ACCEPTANCE (Fischer's exact test)

	Not Satisfied	Satisfied	Good	Excellent	p value
Group D	4(16%)	14(56%)	4 (16%)	3 (12%)	0.0002
Group C	18 (72%)	5 (20%)	2 (8%)	0 (0%)	

The patient acceptance of the study drug in Group D found to be not satisfied, satisfied ,good and excellent were 16%,56%,16% and 12% respectively when compared to Group C were 72%,20%,8% and 0% respectively. There was significant statistical difference between the two groups in the patient acceptance ($p<0.05$)



DISCUSSION

In human beings, studies using epidural α-2 agonists like clonidine and dexmedetomidine have been conducted without any report of neurological deficit. To avoid neuraxial opioid induced side effects such as respiratory depression, nausea, urinary retention and pruritus.[32-34] α-2 agonists are being extensively evaluated as an alternative for post operative pain relief. Epidural administration of α-2 agonists is associated with sedation, analgesia, anxiolysis, hypnosis and sympatholysis.[3-4] Clonidine has been used successfully over the last decade for the above said purpose and the introduction of dexmedetomidine has further widened the scope of α-2 agonists in post operative epidural analgesia. Faster onset of action of local anaesthetics, prolonged duration of post operative analgesia and stable cardiovascular parameters makes these agents very effective adjuvants in post operative analgesia.[12, 19, 25]

In our study 2µg/kg of dexmedetomidine (made upto 3ml with normal saline) was added to 7ml of 0.125% bupivacaine or 2µg/kg of clonidine (made upto 3ml with normal saline) added to 7ml of 0.125% bupivacaine and its efficacy as an adjuvant in post operative epidural analgesia was studied in 50 patients who underwent elective upper abdominal surgery.

The demographic profile of our patients in both groups was comparable with respect to mean age, body weight, ASA Physical status.

Duration of analgesia:

The results of the study have shown that addition of either 2μg/kg dexmedetomidine or 2μg/kg clonidine as an adjuvant to epidural bupivacaine (0.125%) not only prolongs the duration of analgesia but also provide a good sedation level in the post operative period. Dexmedetomidine has a visible edge over clonidine as it enables an earlier onset and establishment of analgesia. The addition of these two adjuvants promotes faster onset compared to established time of onset of sensory analgesia with bupivacaine alone.

This was correlated with the studies done by

1. Antonio Mauro Vieira, TSA, M.D [38], et.al on epidural dexmedetomidine or clonidine in post cholecystectomy analgesia and sedation. There has been analgesia in both groups, especially at 2 and 6 hours. There has been statistically significant difference among periods of 2, 6 and 24 hours in the dexmedetomidine group; in the clonidine group, this statistically significant difference was observed between the periods of 2 and 6 hours and between 2 and 24 hours.

2. El-Henaway AM [12] et.al on addition of clonidine or dexmedetomidine to bupivacaine prolongs caudal analgesia in children. They administered

dexmedetomidine and clonidine, both in a dose of 2μg/kg as an adjuvant with 0.25% bupivacaine caudally. They found that the duration of analgesia was significantly higher in the group receiving bupivacaine-dexmedetomidine mixture (median 95%CI): 16 hours or bupivacaine-clonidine mixture (median 95% CI): 5 hours.

3. Neogi [39] et.al on a comparative study between clonidine and dexmedetomidine used as adjuvants to ropivacaine for caudal analgesia in paediatric patients. They compared clonidine 1μg/kg and dexmedetomidine 1μg/kg as adjuvants to ropivacaine 0.25% for caudal analgesia in paediatric patients. The mean duration of analgesia was 6.32±0.46 hours in ropivacaine, 13.17±0.68 hours in clonidine group and 15.26±0.86 hours in dexmedetomidine group.

4. Saadawy I [13], et.al compared caudal bupivacaine 0.25% administered with dexmedetomidine 1μg/kg and caudal bupivacaine alone. The duration of analgesia was significantly longer with dexmedetomidine administration ($p<0.001$)

Sedation score:

The results of our study clearly indicates the sedation score between the two groups was similar in the first two hours after study drug administration and they had profound sedation but arousable by gentle tactile stimulation(i.e.

Ramsay sedation score of 3). After two hours, the percentage of dexmedetomidine group patients who have scored higher sedation scores is more compared to clonidine group. But the patients remained awake but calm in both the groups (Score 2). Overall the sedation scores were highly significant statistically with administration of dexmedetomidine. [11, 26, 27]

This was correlated with the study done on Dexmedetomidine and clonidine in epidural anaesthesia: A comparative evaluation by Sukhminder Jit Singh Bajwa [42] et.al.They found that epidural dexmedetomidine produced profound sedation in 36% of the patients who were arousable by gentle tactile stimulation compared to achievement of similar sedation level in 16% of the patients in clonidine group. Thirty two percent of the patients remained awake but calm in clonidine group compared to 16% in dexmedetomidine group who were equally cooperative and calm.

Time to Rescue Analgesia:

The group D showed comparatively good results over group C in various characteristics like prolonged post operative analgesia and a lesser amount of rescue analgesia used post operatively. The mean time for rescue analgesia is more in Group D (425.6±64.27 minutes) when compared to Group C (226±24.83 minutes)

This was correlated with the study done by Sukhminder Jit Singh Bajwa [42] et al.They administered 17ml of 0.75% epidural ropivacaine and 1.5µg/kg of dexmedetomidine to Group RD while Group RC received 17ml of 0.75% epidural ropivacaine and 2µg/kg of clonidine. The time for rescue analgesia was comparatively shorter (310.76±23.75) in the patients who were administered clonidine ($P<0.05$)

Hemodynamic stability:

The vital signs remained stable throughout the study period which confirms the established effects of α-2 agonists in providing a hemodynamically stable post operative period. Although a slight decrease in heart rate and blood pressure (both systolic and diastolic) was observed in both the groups, it never fell down to more than 20% of baseline values. But hypotension and bradycardia were observed more in clonidine group patients.

This was correlated with the study done by Sukhminder Jit Singh Bajwa [42], et.al on Dexmedetomidine and clonidine in epidural anaesthesia- A comparative evaluation.

Side effects:

The side effect profile of both the drugs was quite favourable as none of the patient in either group had profound deep sedation or respiratory depression which correlates very well with other studies.

We observed a little higher incidence of dry mouth, nausea, vomiting and dizziness in clonidine group than dexmedetomidine group. This was not correlated with the study done by Sukhminder Jit Singh Bajwa [42], et.al on Dexmedetomidine and clonidine in epidural anaesthesia-A comparative evaluation.

We observed from our study that the duration of postoperative analgesia recorded a mean of 417.32±67.36 minutes in Group D with 217.2±25.32 minutes in Group C, with a P-value of 0.0001.Group D patients achieved a statistically significant higher sedation score compared with Group C patients. The post-op hemodynamic variables between the groups were comparable and were statistically significant. The results of our observations show that in addition to prolonged analgesia, dexmedetomidine has a favourable safety profile and stable hemodynamics over clonidine, which correlates with the reports published by other authors.

SUMMARY

This single blinded prospective randomized controlled study was done to evaluate the duration of analgesia as well as sedation and adverse effects of dexmedetomidine(2μg/kg) vs. clonidine(2 μg/kg) given via epidural route with 0.125% bupivacaine in patients post operatively who underwent upper abdominal surgery under general anaesthesia.

The following observations were made:

1. The addition of dexmedetomidine to 0.125% bupivacaine significantly prolonged the duration of post operative analgesia.

2. The addition of dexmedetomidine significantly prolonged the time for demand analgesia. Most of the patients who received dexmedetomidine did not receive 2nd rescue analgesia in the first 24 hours of post operative period.

3. The addition of dexmedetomidine epidurally produced profound sedation that was arousable for many hours compared to clonidine.

4. The incidence of side effects such as hypotension, bradycardia and vomiting are more in patients who received clonidine which was not statistically significant between the groups.

5. No episode of respiratory depression was noted in both the study groups which are more common with opioids.

6. With regard to acceptance, the patients who received dexmedetomidine were highly satisfied by the study drug administration.

CONCLUSION

To conclude that 2 µg/kg of dexmedetomidine seems to be a better adjuvant to epidural bupivacaine (0.125%) in post operative analgesia. It has excellent quality of post operative analgesia and a prolonged duration of arousable sedation with minimal side effects. The hemodynamic stability was well maintained with dexmedetomidine. Overall the experience with dexmedetomidine was quite satisfactory as compared to clonidine because of its superior sedative, anxiolytic properties and patient comfortability.

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INFORMATION TO PARTICIPANTS

Investigation : Dr.Swarnalingam.T

Name of the Participant :

Title : Prospective, Randomized comparative study on effect of adding clonidine Vs Dexmeditomidine to epidural bupivacaine (0.125%) on post operative analgesia in upper abdominal surgeries.

You are invited to take part in this research study. The information in this document is meant to help. The information in this document is meant to help you decide whether or not to take part. Please feel free to ask if you have any queries or concerns.

You are being asked to participate in this study being conducted in the Institute of Anaesthesiology and Critical care, Madras Medical College.

What is the Purpose of the Research

For upper abdominal surgeries, epidural analgesia administered with the drug 0.125% or 0.0625% Bupivaicane with or without adjuvants (like Fentanyl). This gives the pain relief for a reasonable period of time. In this study adding clonidine Vs Dexmeditomidine (selective alpha 2 agonists) to Epidural 0.125% Bupivacaine and its effects on post operative analgesia.

The Study Design

All the patients in the study will be divided into two groups. One group will receive epidural dexmeditomidine and another group will receive epidural clonidine after the surgery. In both the group surgery will be done under general anaesthesia.

Benefits

By review of previous study post operative analgesia after epidural dexmeditomidine or clonidine will be prolonged. Both drug also has sedative properly which reduces the conception of other sedative drugs.

Discomforts and risks

Hypotension and Bradycardia- common side effects of sedative $\alpha 2$ agonists. Rarely nausea and vomiting also occur. Inj.Ondensetron will be given for vomiting. Hypotension and Bradycardia will be treated with Inj.Ephedrine, Inj.Atropine respectively.

And if you do not want to participate you will have alternative of setting the standard treatment and your safety is our prime concern.

Time :
Date :
Place : Signature / Thumb Impression of Patient
Patient Name:

Signature of the Investigator : _____

Name of the Investigator : _____

PROFORMA

"PROSPECTIVE, RANDOMIZED COMPARATIVE STUDY ON EFFECT OF ADDING CLONIDINE vs DEXMEDITOMIDINE TO EPIDURAL BUPIVACAINE (0.125%) ON POST OPERATIVE ANALGESIA IN UPPER ABDOMINAL SURGERIES".

NAME: AGE: SEX: I.P.No:

DIAGNOSIS: SURGERY PLANNED:

PREOPERATIVE ASSESSMENT:

HISTORY:

CO-MORBID ILLNESS & TREATMENT DETAILS:

EFFORT TOLERANCE- _____ METS

H/O PREVIOUS SURGERY:

GENERAL EXAMINATION:

HEIGHT: WEIGHT: BMI: ANAEMIA- JAUNDICE -

PULSE- BP- CVS- RS-- SPINE-

INVESTIGATIONS:

BT: CT: BLOOD GROUPING&TYPING:

BLOOD SUGAR: UREA: CREATININE: ECG: CXR:

EPIDURAL CATHETERISATION:

POSITION SPACE NEEDLE SIZE

MATERIALS:

EPIDURAL NEEDLE -- TUOHY NEEDLE 16/18 G

OUTCOME MEASURES:

QUALITY OF ANALGESIA USING VAS SCORE

LEVEL OF SEDATION USING RAMSAY SEDATION SCORE

HEART RATE, BLOOD PRESSURE

DATA ANALYSIS: USING STATISTICAL PACKAGE

POST- OP VITAL PARAMETERS:

TIME	HR	SBP	DBP	SPO2	RR	VAS SCORE	RSS SCORE

SIDE EFFECTS:

HYPOTENSION

BRADYCARDIA

NAUSEA

VOMITING

DIZZINESS

RESPIRATORY DEPRESSION

“A PROSPECTIVE, RANDOMIZED COMPARATIVE STUDY ON EFFECT OF ADDING CLONIDINE vs DEXMEDETOMIDINE TO EPIDURAL BUPIVACAINE (0.125%) ON POSTOPERATIVE ANALGESIA IN UPPER ABDOMINAL SURGERIES”

ABSTRACT

Introduction

Effective pain control is essential for optimal care of surgical patients. The use of epidural analgesia for the management of post operative pain has evolved as a critical component of multi modal approach to achieve the role of adequate analgesia with improved outcome. Epidural analgesia offers superior post operative pain relief compared with systemic drugs. In addition to improved pain control, epidural analgesia can improve patient outcome by attenuating detrimental post operative stress

Keeping their pharmacologic interactions and other properties we planned to conduct a single blinded prospective randomized clinical controlled study at our institution in patients who underwent upper abdominal surgery under general anaesthesia with an aim to compare the analgesic and sedative effect of these drugs given via epidural route as an adjuvant to 0.125% bupivacaine in the post operative period.

Aims & Objectives:

To compare the efficacy and safety of two α-2 adrenergic agonists, Dexmedetomidine and Clonidine in post-operative epidural analgesia after upper abdominal surgery with respect to their sedative properties and hemodynamic stability.

Methodology:

50 ASA I & II patients aged 18-60 years undergoing upper abdominal surgery under general anaesthesia and planned for an epidural analgesia post operatively. Epidural catheter tip was kept around T6 -T8 level.Patients will be randomly selected to 2 groups.

Group C: 7ml of 0.125% bupivacaine + 2ug/kg clonidine made to 3 ml with normal saline.

Group D: 7 ml of bupivacaine + 2 ug/kg of dexmeditomidine made to 3 ml with normal saline.

Drug was injected via epidural catheter just before extubation. Patients were shifted to PACU for observation. Various parameters like HR, MAP, SPo2, and VAS are observed for 24 hours post operatively. Incidences of side effects are also noted. Injection Tramadol 50 mg IV is used as rescue analgesia when VAS ≥4.

Observation & Results:

The mean duration of analgesia was 417.32±67.36 minutes in Group D and 217.2±25.32 minutes in Group C. There was statistically significant difference among two groups in the mean duration of analgesia ($P<0.05$).

The mean time for 1st rescue analgesia was 425.6±64.27 minutes in Group D and 226±24.83 minutes in Group C. There was significant difference among two groups in the duration of time for rescue analgesia (P<0.05).

There was no difference in pain score at 0,30 and 120 minutes and was found to be statistically not significant (p>0.05). At 360 minutes, the mean VAS score in Group D was 0.84±0.89 and in Group C was 1.76±0.99; there was statistical significant difference in both groups (p<0.05)**.** The mean VAS score in Group D was 2.96±1.01 and in Group C was 2.08±1.07 at 720 minutes which was found to be statistically significant (p<0.05)**.** At 1440 minutes, the mean VAS score in Group D was 3.48±0.82 and in Group C was 3.52±1.04 and was found to be statistically not significant (p>0.05).

Conclusion:

To conclude that 2 μg/kg of dexmedetomidine seems to be a better adjuvant to epidural bupivacaine (0.125%) in post operative analgesia. It has excellent quality of post operative analgesia and a prolonged duration of arousable sedation with minimal side effects. The hemodynamic stability was well maintained with dexmedetomidine. Overall the experience with dexmedetomidine was quite satisfactory as compared to clonidine because of its superior sedative, anxiolytic properties and patient comfortability.

MASTER CHART

DEXMEDETOMIDINE

S.NO	Name	Age	Sex	Weight	Procedure	ASA-PS	Duration of Analgesia (in minutes)	Time to 1st Rescue Analgesia (in minutes)	Time to 2nd Rescue Analgesia (in minutes)
1	PURUSHOTHAMAN	49	M	63	OPEN CHOLE	II	425	435	NIL
2	SARAVANAN	48	M	58	OPEN CHOLE	II	485	490	NIL
3	ARUMUGAM	42	M	56	OPEN CHOLE	I	420	430	NIL
4	KUMAR	36	M	55	LAPARATOMY	I	380	390	890
5	SIVA	60	M	67	LAPARATOMY	II	368	380	780
6	INDIRANI	45	F	65	TV &GJ	I	350	355	855
7	KALPANA	31	F	48	OPEN CHOLE	I	380	390	870
8	KANCHANA	35	F	46	OPEN CHOLE	I	390	395	910
9	VIJAYA	58	M	55	OPEN CHOLE	II	425	430	NIL
10	KRISHNAVENI	27	F	55	MARSUPILISATION	I	405	415	NIL
11	KUPPAN	60	M	67	OPEN CHOLE	II	410	425	NIL
12	ZORA BEGUM	29	F	54	OPEN CHOLE	I	280	300	710
13	MATHAN	36	M	59	OPEN CHOLE	I	315	330	740
14	HARI	53	M	56	OPEN CHOLE	I	420	430	810
15	ASHOKAN	45	M	64	LAPARATOMY	I	485	490	NIL
16	PERUMAL	58	M	59	LAPARATOMY	II	490	495	NIL
17	PELGISH	44	F	57	OPEN CHOLE	I	540	545	NIL
18	NAGARAJ	46	M	63	OPEN CHOLE	I	520	525	NIL
19	DILLIKUMAR	43	M	68	OPEN CHOLE	I	505	510	NIL
20	SHEHIM BEGUM	37	F	56	OPEN CHOLE	I	485	490	NIL
21	AKKAIAH	40	M	67	JC ANASTOMOSIS	I	390	395	910
22	RAMACHANDRAN	59	M	62	CBD EXPLORATION	II	425	430	NIL
23	VELMURUGAN	34	M	58	OPEN CHOLE	I	440	445	NIL
24	SIVAGAMI	21	F	45	LAPARATOMY	II	405	410	NIL
25	VASANTHA	22	F	50	CBD EXPLORATION	I	295	310	NIL

TV & GJ- TRUNCAL VAGOTOMY & GASTROJEJUNOSTOMY

OPEN CHOLE- OPEN CHOLECYSTECTOMY, JC ANASTOMOSIS- JEJUNOCOLIC ANASTOMOSIS

CLONIDINE

S.NO	Name	Age	Sex	Weight	Procedure	ASA-PS	Duration of Analgesia (in minutes)	Time to 1st Rescue Analgesia (in minutes)	Time to 2nd Rescue Analgesia (in minutes)
1	VENUGOPAL	55	M	55	OPEN CHOLE	II	220	225	725
2	KANDASAMY	40	M	65	LAPARATOMY	I	185	195	655
3	SUBRAMANI	35	M	59	OPEN CHOLE	I	210	215	815
4	SERINA	35	F	53	OPEN CHOLE	I	240	250	850
5	SELVAM	46	M	66	CYSTOGASTROTOMY	I	220	230	930
6	SUMATHI	29	F	54	LAPARATOMY	I	215	220	830
7	VARADHAN	32	M	58	GJ	I	195	210	710
8	SARAN	40	M	52	CHOLEDO	I	185	195	795
9	SEKAR	45	M	68	OPEN CHOLE	II	220	230	810
10	PANCHATCHARAM	55	M	65	OPEN CHOLE	II	280	290	NIL
11	MUUNIYAMMAL	45	F	64	LAPARATOMY	I	290	295	NIL
12	VIMALA	50	F	55	OPEN CHOLE	II	185	200	705
13	ARUMUGAM	48	M	62	OPEN CHOLE	I	210	220	855
14	KUMAR	48	M	56	TV&GJ	I	220	225	840
15	ADHILAKSHMI	53	F	48	OPEN CHOLE	I	225	235	825
16	VENGAIAH	61	M	61	OPEN CHOLE	II	220	230	785
17	CHANDRA	45	F	54	OPEN CHOLE	I	205	215	825
18	DEVARAJ	40	M	46	TV&GJ	II	200	210	760
19	PURUSHOTHAMAN	49	M	67	OPEN CHOLE	I	195	210	720
20	SARAVANAN	48	M	54	OPEN CHOLE	I	210	215	850
21	MOHAMMED KADAR	46	M	65	OPEN CHOLE	I	225	230	655
22	MINNALA	45	F	68	OPEN CHOLE	II	245	255	760
23	RAMESH	30	M	61	OPEN CHOLE	I	195	205	720
24	MURUGAN	45	M	59	TV&GJ	I	210	215	755
25	THANGAVELU	57	M	65	LAPARATOMY	II	225	230	835

TV & GJ- TRUNCAL VAGOTOMY & GASTROJEJUNOSTOMY

CHOLEDO- CHOLEDOCHOJEJUNOSTOMY

OPEN CHOLE- OPEN CHOLECYSTECTOMY

DIASTOLIC BLOOD PRESSURE – DEXMEDETOMIDINE

S No	15 MIN	30 MIN	120 MIN	240 MIN	360 MIN	480 MIN	600 MIN	720 MIN	840 MIN	96O MIN	1080 MIN	1200 MIN	1320 MIN	1440 MIN
1	70	69	67	66	65	65	69	66	65	68	65	68	66	66
2	65	64	63	58	64	61	61	67	62	63	63	60	62	62
3	66	66	68	65	68	65	66	69	65	65	66	67	68	70
4	70	71	67	68	69	69	70	70	66	69	68	69	71	69
5	70	69	66	67	65	65	66	69	68	65	68	65	66	66
6	66	66	65	64	64	66	65	64	63	64	65	66	63	65
7	70	67	69	68	69	65	69	68	69	66	68	70	69	68
8	65	63	66	65	64	63	64	63	64	65	66	64	65	66
9	68	69	70	68	66	69	68	69	65	69	68	69	67	70
10	60	62	61	60	61	60	61	61	60	59	57	60	60	50
11	50	60	60	57	59	60	61	61	60	61	60	61	62	60
12	70	68	69	66	68	69	68	66	68	68	69	68	68	66
13	65	67	66	66	68	67	67	66	66	65	63	63	63	66
14	64	63	63	63	65	65	64	66	64	64	63	63	64	64
15	68	68	69	67	66	69	68	68	68	66	68	69	69	68
16	57	57	57	59	60	60	57	59	59	58	58	59	58	59
17	66	66	68	65	66	67	69	67	65	65	64	66	66	66
18	63	63	62	62	62	63	63	63	62	61	61	63	62	62
19	64	64	64	64	66	65	65	63	64	64	64	65	64	64
20	63	63	65	64	66	66	63	63	65	64	63	63	63	64
21	60	62	62	61	61	62	60	63	62	61	60	59	60	59
22	63	62	62	61	63	62	66	63	60	60	60	61	61	60
23	65	64	64	64	59	58	59	64	63	58	59	63	64	64
24	59	60	59	63	60	60	61	61	63	62	63	62	58	58
25	60	59	68	60	59	60	61	59	60	61	63	60	59	59

DIASTOLIC BLOOD PRESSURE – CLONIDINE

S No	15 MIN	30 MIN	120 MIN	240 MIN	360 MIN	480 MIN	600 MIN	720 MIN	840 MIN	96O MIN	1080 MIN	1200 MIN	1320 MIN	1440 MIN
1	59	58	59	59	60	58	59	65	62	60	61	62	60	60
2	64	62	61	63	64	62	60	61	63	61	64	62	65	61
3	58	58	59	60	58	56	56	57	55	58	60	63	64	59
4	68	69	66	67	66	69	68	66	65	70	68	66	68	69
5	68	69	66	67	66	69	68	66	65	70	68	66	68	69
6	62	61	60	62	64	60	61	63	62	64	60	59	66	61
7	55	56	57	55	58	59	60	54	55	60	55	58	59	60
8	60	61	60	63	61	64	62	61	60	62	65	60	61	62
9	60	61	60	63	61	64	62	61	60	62	65	60	61	62
10	62	60	60	62	61	63	61	61	62	60	60	62	60	62
11	61	62	60	60	61	63	60	63	60	61	60	62	60	61
12	68	62	66	60	61	62	60	62	60	61	66	60	62	61
13	62	61	62	60	61	62	62	61	60	59	60	63	61	61
14	60	60	61	62	63	62	60	62	60	60	61	62	60	61
15	60	61	61	60	60	61	62	61	61	60	61	62	61	61
16	60	62	61	69	60	63	61	62	61	61	61	64	60	61
17	59	59	55	59	60	59	58	59	59	60	60	59	64	59
18	62	62	61	60	62	61	64	63	62	61	60	61	60	60
19	62	61	62	60	63	64	65	61	62	65	61	60	60	63
20	59	59	58	60	56	55	56	57	58	59	60	61	62	60
21	62	62	64	61	62	60	66	68	61	60	61	60	61	63
22	60	62	64	66	68	61	62	68	60	61	61	60	61	60
23	59	60	58	57	58	60	56	57	58	55	58	59	64	63
24	60	61	63	62	62	60	62	61	66	65	60	60	61	61
25	61	62	64	60	61	61	60	61	61	65	60	62	62	61

PULSE RATE- DEXMEDETOMIDINE

S No	15 MIN	30 MIN	120 MIN	240 MIN	360 MIN	480 MIN	600 MIN	720 MIN	840 MIN	96O MIN	1080 MIN	1200 MIN	1320 MIN	1440 MIN
1	58	56	56	57	58	59	59	58	58	56	59	57	58	60
2	59	61	60	60	61	62	62	59	63	61	61	62	60	60
3	65	64	67	66	65	68	65	69	65	65	66	66	67	64
4	58	56	57	57	55	59	57	58	57	56	57	58	58	59
5	64	64	63	63	62	61	66	63	60	62	63	61	60	63
6	55	58	58	47	49	54	55	49	57	56	52	55	55	54
7	66	66	65	67	67	64	64	63	63	63	63	63	64	62
8	66	66	65	67	66	63	66	67	66	65	66	65	67	68
9	59	58	57	60	58	57	58	57	59	57	56	58	57	58
10	58	58	59	60	59	59	58	57	59	59	59	58	59	59
11	67	66	69	70	71	73	69	68	68	69	69	69	68	67
12	66	66	67	68	68	67	67	66	68	66	66	67	67	66
13	77	76	76	75	74	76	79	71	69	72	71	77	76	75
14	70	70	71	71	71	72	71	70	71	70	72	73	71	71
15	67	66	68	69	68	68	69	70	69	69	68	69	69	65
16	54	55	49	49	54	52	52	53	54	55	49	48	55	53
17	58	60	59	59	58	58	57	59	59	58	57	58	58	56
18	57	60	59	58	59	56	57	59	58	59	58	58	58	58
19	59	59	60	58	59	59	57	60	63	59	58	59	60	59
20	67	63	63	60	60	59	62	61	58	62	60	60	61	61
21	66	63	62	61	60	58	60	62	60	64	62	58	61	60
22	59	59	58	58	60	61	61	62	62	59	60	61	62	60
23	68	69	66	67	69	66	67	66	69	66	65	70	68	62
24	67	66	66	65	65	65	66	59	64	64	64	62	60	61
25	57	59	59	60	60	60	61	60	61	61	58	58	56	59

PULSE RATE – CLONIDINE

S No	15 MIN	30 MIN	120 MIN	240 MIN	360 MIN	480 MIN	600 MIN	720 MIN	840 MIN	96O MIN	1080 MIN	1200 MIN	1320 MIN	1440 MIN
1	56	55	56	57	57	53	56	54	55	56	55	56	56	57
2	51	47	49	53	55	53	52	51	53	55	50	51	52	54
3	58	57	58	59	58	59	57	57	57	59	58	60	59	59
4	66	69	69	68	68	67	68	67	68	68	66	68	65	68
5	65	64	63	62	60	60	60	63	63	60	60	63	62	63
6	63	63	63	63	62	62	61	63	62	60	62	63	63	62
7	55	55	54	56	56	55	54	56	55	55	54	54	55	56
8	56	56	58	58	58	57	57	55	55	58	57	57	56	56
9	56	55	55	59	54	55	56	59	59	55	57	58	58	58
10	66	63	61	62	62	66	60	60	62	62	62	60	61	58
11	69	70	69	68	67	71	74	73	70	69	68	70	69	71
12	57	58	59	60	58	58	57	59	58	58	58	59	59	59
13	52	52	51	55	51	51	52	52	51	55	53	52	52	51
14	59	60	58	55	58	57	60	59	59	58	60	57	58	58
15	57	58	59	59	60	60	59	58	59	59	57	60	59	59
16	51	48	49	55	54	57	52	52	54	51	50	54	52	51
17	62	60	60	62	62	62	60	62	60	64	61	63	63	63
18	46	49	53	55	53	55	57	52	52	55	55	55	53	59
19	53	55	48	56	55	54	49	53	49	51	52	46	55	55
20	64	63	62	62	63	61	61	62	63	59	64	63	62	62
21	70	71	70	69	72	73	73	71	72	70	72	70	69	70
22	57	59	58	58	57	59	60	59	59	59	59	58	59	58
23	60	62	61	63	64	62	63	60	59	62	61	61	63	61
24	64	63	63	62	60	60	61	62	62	61	62	62	59	62
25	59	60	60	62	56	50	59	61	62	61	59	53	59	61

SYSTOLIC BLOOD PRESSURE- DEXMEDETOMIDINE

S No	15 MIN	30 MIN	120 MIN	240 MIN	360 MIN	480 MIN	600 MIN	720 MIN	840 MIN	96O MIN	1080 MIN	1200 MIN	1320 MIN	1440 MIN
1	110	113	110	112	112	115	118	110	113	112	119	115	113	112
2	103	100	107	103	102	105	105	102	103	104	103	102	104	103
3	109	110	107	110	112	11	110	109	108	114	110	112	110	112
4	123	126	123	134	125	124	127	125	127	126	127	128	126	128
5	117	116	115	117	115	118	116	115	115	118	116	117	118	118
6	110	112	109	108	109	110	110	112	110	109	109	108	109	110
7	108	108	108	110	112	114	110	109	112	110	114	112	110	109
8	108	110	106	112	108	106	112	119	104	108	107	110	108	106
9	105	105	104	103	104	105	109	110	106	108	102	100	101	100
10	89	92	90	93	91	93	91	93	90	92	91	93	91	92
11	100	102	102	101	103	100	103	102	101	103	103	102	102	101
12	119	118	117	118	120	117	116	119	116	118	118	120	117	120
13	113	114	116	115	115	114	116	115	117	112	111	112	113	114
14	115	112	115	116	116	115	115	116	114	113	114	115	115	114
15	108	107	104	110	108	103	100	103	102	105	104	105	106	105
16	98	90	95	91	98	92	89	90	91	93	94	91	89	88
17	100	103	106	102	104	103	104	102	103	102	101	100	102	100
18	100	104	98	109	100	103	100	108	102	101	103	102	100	100
19	102	103	99	103	102	101	105	103	101	103	102	103	101	100
20	101	101	110	104	103	112	112	102	100	100	102	102	101	102
21	108	107	104	107	108	108	109	100	112	112	110	108	106	106
22	112	112	110	111	115	113	110	114	112	110	113	110	110	110
23	110	107	109	108	119	110	110	110	108	106	109	105	110	107
24	108	110	110	109	106	108	112	115	109	106	104	110	112	117
25	110	112	117	111	112	116	114	113	115	115	114	105	104	104

SYSTOLIC BLOOD PRESSURE – CLONIDINE

S No	15 MIN	30 MIN	120 MIN	240 MIN	360 MIN	480 MIN	600 MIN	720 MIN	840 MIN	96O MIN	1080 MIN	1200 MIN	1320 MIN	1440 MIN
1	89	88	88	89	86	89	86	85	87	90	89	99	100	100
2	102	102	100	103	101	101	104	102	104	101	102	103	103	102
3	89	99	88	90	88	90	89	88	87	90	89	100	99	99
4	108	105	105	105	104	104	103	104	104	105	104	104	104	104
5	105	104	104	104	104	105	105	105	102	105	104	106	103	103
6	100	102	101	103	100	100	103	102	101	99	100	101	102	101
7	90	88	90	89	91	90	91	88	90	90	87	100	102	102
8	99	99	100	101	102	101	99	100	99	98	100	99	98	100
9	101	102	100	101	103	102	103	100	100	102	102	102	100	103
10	104	103	103	105	104	105	105	104	104	103	103	103	105	104
11	104	105	103	103	103	102	104	104	105	104	105	103	103	104
12	100	103	102	102	104	103	100	100	102	103	102	103	102	103
13	103	102	103	102	103	103	102	100	100	103	104	103	100	103
14	104	104	105	106	104	104	105	106	105	104	104	100	110	104
15	102	103	102	102	102	103	104	102	103	110	110	112	114	115
16	106	104	107	108	108	106	110	109	108	108	109	110	106	109
17	89	90	88	86	90	90	92	99	102	100	105	106	100	110
18	100	102	100	108	110	109	106	110	109	102	100	104	100	110
19	109	102	105	103	103	102	107	103	108	108	110	102	102	110
20	98	99	88	90	88	87	89	89	99	90	89	100	108	106
21	103	103	104	102	109	108	105	105	109	110	109	106	105	100
22	99	109	110	109	109	107	108	103	109	110	111	117	118	110
23	99	98	96	89	88	90	89	88	100	98	109	100	110	109
24	103	102	104	107	104	109	110	105	100	108	100	100	109	108
25	103	104	102	103	105	104	103	102	105	106	102	103	105	102

VISUAL ANALOGUE SCORE – DEXMEDETOMIDINE

S No	15 MIN	30 MIN	120 MIN	240 MIN	360 MIN	480 MIN	600 MIN	720 MIN	840 MIN	96O MIN	1080 MIN	1200 MIN	1320 MIN	1440 MIN
1	0	0	0	2	3	3	3	3	3	3	3	3	3	3
2	0	0	0	2	3	3	3	3	3	3	3	3	3	3
3	0	0	0	2	3	3	3	3	3	3	3	3	3	3
4	0	0	0	4	3	3	4	4	4	4	4	4	4	4
5	0	0	0	4	3	3	4	4	4	4	4	4	4	4
6	0	0	0	4	3	3	4	4	4	4	4	4	4	4
7	0	0	0	4	3	3	4	4	4	4	4	4	4	4
8	0	0	0	3	3	3	4	4	4	4	4	4	4	4
9	0	0	0	2	2	3	3	3	3	3	3	3	3	3
10	0	0	0	2	2	3	3	3	3	3	3	3	3	3
11	0	0	0	2	2	2	3	3	3	3	3	3	3	3
12	0	0	0	4	3	4	4	4	4	4	4	4	4	4
13	0	0	0	4	3	4	4	4	4	4	4	4	4	4
14	0	0	0	4	3	4	4	4	4	4	4	4	4	4
15	0	0	0	2	2	3	3	3	3	3	3	3	3	3
16	0	0	0	1	2	3	2	2	2	2	2	2	2	2
17	0	0	0	1	2	3	2	2	2	2	2	2	2	2
18	0	0	0	1	2	2	2	2	2	2	2	2	2	2
19	0	0	0	1	2	2	2	2	2	2	2	2	2	2
20	0	0	0	1	2	3	2	2	2	2	2	2	2	2
21	0	0	0	1	2	3	2	2	2	3	3	3	3	3
22	0	0	0	3	3	3	4	4	4	4	4	4	4	4
23	0	0	0	2	3	3	3	3	3	3	3	3	3	3
24	0	0	0	2	3	3	3	3	3	3	3	3	3	3
25	0	0	0	2	3	3	3	3	3	3	3	3	3	3

VISUAL ANALOGUE SCORE – CLONIDINE

S No	15 MIN	30 MIN	120 MIN	240 MIN	360 MIN	480 MIN	600 MIN	720 MIN	840 MIN	96O MIN	1080 MIN	1200 MIN	1320 MIN	1440 MIN
1	0	0	0	4	2	3	3	3	3	3	3	4	4	4
2	0	0	0	4	3	3	4	3	4	4	4	4	4	4
3	0	0	0	4	2	3	3	3	3	3	3	4	4	4
4	0	0	0	4	2	3	3	3	3	3	4	4	4	4
5	0	0	0	4	2	3	3	3	3	3	3	4	4	4
6	0	0	0	4	2	2	3	3	3	3	4	4	4	4
7	0	0	0	4	2	2	2	2	3	3	3	3	4	4
8	0	0	0	4	3	3	3	3	3	3	4	4	4	4
9	0	0	0	4	3	3	3	3	3	4	4	4	4	4
10	0	0	0	4	2	2	2	2	3	3	4	4	4	4
11	0	0	0	4	2	2	2	2	3	3	3	4	4	4
12	0	0	0	4	2	2	2	2	3	3	4	4	4	4
13	0	0	0	4	3	3	3	3	3	4	4	4	4	4
14	0	0	0	4	3	3	3	3	4	4	4	4	4	4
15	0	0	0	4	2	2	3	3	3	4	4	4	4	4
16	0	0	0	4	2	2	3	3	3	3	3	4	4	4
17	0	0	0	4	2	3	3	3	3	4	4	4	4	4
18	0	0	0	4	2	3	3	3	3	4	4	4	4	4
19	0	0	0	4	3	3	3	3	3	3	4	4	4	4
20	0	0	0	4	3	3	3	3	3	3	4	4	4	4
21	0	0	0	4	2	3	3	3	3	3	4	4	4	4
22	0	0	0	4	2	3	4	2	3	3	3	4	4	4
23	0	0	0	4	2	3	4	2	3	3	4	4	4	4
24	0	0	0	4	3	3	4	2	3	3	4	4	4	4
25	0	0	0	4	2	3	4	2	3	3	4	4	4	4

SPO2 DEXMEDETOMIDINE

S No	15 MIN	30 MIN	120 MIN	240 MIN	360 MIN	480 MIN	600 MIN	720 MIN	840 MIN	96O MIN	1080 MIN	1200 MIN	1320 MIN	1440 MIN
1	99%	99%	98%	98%	99%	99%	98%	99%	99%	98%	99%	98%	99%	98%
2	99%	99%	99%	99%	99%	99%	99%	98%	99%	98%	99%	99%	98%	99%
3	99%	99%	99%	98%	99%	98%	99%	98%	99%	98%	98%	99%	99%	99%
4	100%	100%	98%	99%	99%	99%	99%	98%	97%	97%	99%	98%	99%	99%
5	99%	99%	98%	98%	98%	99%	99%	99%	97%	98%	99%	99%	99%	99%
6	98%	99%	99%	99%	99%	99%	99%	98%	99%	99%	99%	99%	98%	99%
7	99%	99%	99%	98%	99%	99%	99%	99%	99%	99%	100%	99%	99%	100%
8	100%	100%	100%	99%	99%	99%	99%	99%	99%	98%	99%	99%	99%	99%
9	99%	99%	99%	99%	98%	99%	98%	99%	99%	99%	98%	99%	99%	99%
10	99%	99%	99%	99%	98%	98%	99%	99%	99%	99%	99%	99%	98%	99%
11	97%	99%	99%	99%	99%	99%	99%	99%	99%	98%	99%	99%	99%	99%
12	99%	99%	98%	98%	99%	99%	99%	99%	98%	98%	99%	97%	99%	99%
13	99%	99%	100%	100%	99%	99%	98%	99%	98%	99%	99%	99%	99%	99%
14	98%	99%	99%	99%	99%	98%	98%	99%	99%	99%	99%	99%	99%	99%
15	99%	99%	98%	99%	99%	99%	99%	99%	99%	99%	98%	99%	99%	99%
16	99%	99%	98%	98%	99%	99%	99%	100%	99%	99%	99%	98%	99%	99%
17	99%	99%	99%	99%	98%	98%	99%	99%	99%	99%	99%	99%	99%	99%
18	99%	98%	98%	98%	98%	99%	97%	99%	99%	99%	99%	98%	99%	99%
19	98%	97%	97%	97%	99%	99%	99%	99%	98%	99%	99%	99%	99%	99%
20	99%	99%	99%	99%	98%	98%	98%	98%	99%	99%	99%	99%	99%	99%
21	98%	99%	99%	99%	99%	99%	99%	99%	97%	97%	99%	99%	99%	100%
22	99%	99%	99%	99%	99%	99%	98%	98%	99%	99%	99%	99%	99%	98%
23	99%	99%	99%	99%	98%	98%	99%	99%	99%	97%	98%	99%	99%	99%
24	99%	99%	99%	99%	99%	98%	99%	99%	99%	99%	99%	99%	99%	99%
25	98%	98%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%

SPO2 CLONIDINE

S No	15 MIN	30 MIN	120 MIN	240 MIN	360 MIN	480 MIN	600 MIN	720 MIN	840 MIN	96O MIN	1080 MIN	1200 MIN	1320 MIN	1440 MIN
1	98%	99%	99%	98%	99%	99%	98%	99%	99%	100%	100%	100%	100%	100%
2	98%	99%	99%	99%	99%	99%	99%	99%	99%	100%	100%	100%	100%	100%
3	99%	99%	98%	98%	99%	99%	99%	99%	99%	99%	99%	99%	100%	100%
4	98%	98%	97%	98%	98%	99%	99%	99%	99%	100%	100%	100%	100%	100%
5	99%	99%	99%	99%	99%	100%	100%	100%	100%	100%	100%	100%	100%	100%
6	99%	99%	99%	99%	100%	100%	100%	99%	99%	100%	100%	100%	100%	100%
7	99%	99%	99%	99%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%
8	100%	100%	100%	100%	100%	99%	99%	100%	100%	100%	100%	100%	100%	100%
9	100%	100%	99%	99%	99%	99%	99%	100%	100%	100%	100%	100%	100%	100%
10	99%	99%	99%	99%	99%	99%	99%	100%	100%	100%	100%	100%	100%	100%
11	99%	99%	99%	99%	99%	99%	100%	100%	100%	100%	100%	100%	99%	100%
12	99%	99%	99%	99%	99%	99%	100%	100%	99%	100%	100%	100%	100%	100%
13	99%	99%	99%	99%	100%	100%	100%	100%	100%	100%	100%	100%	100%	100%
14	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	100%	100%
15	100%	100%	100%	99%	99%	99%	99%	99%	99%	100%	100%	100%	100%	100%
16	99%	100%	100%	100%	100%	99%	99%	99%	98%	98%	99%	99%	99%	99%
17	98%	98%	99%	99%	99%	100%	100%	99%	99%	99%	99%	99%	99%	99%
18	98%	98%	99%	98%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%
19	99%	98%	99%	98%	98%	98%	98%	99%	99%	99%	99%	99%	99%	99%
20	99%	99%	99%	99%	99%	99%	98%	98%	98%	98%	99%	99%	99%	99%
21	99%	99%	99%	98%	98%	98%	99%	99%	99%	99%	99%	99%	99%	99%
22	99%	99%	99%	98%	98%	99%	99%	99%	99%	99%	99%	99%	99%	99%
23	98%	99%	99%	98%	98%	99%	99%	98%	99%	99%	99%	99%	99%	99%
24	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%	99%
25	98%	99%	99%	98%	98%	99%	99%	100%	100%	100%	99%	99%	99%	99%

PATIENT ACCEPTANCE

DEXMEDETOMIDINE

S. NO	NOT SATISFIED	SATISFIED	GOOD	EXCELLENT
1	NO	YES	NO	NO
2	NO	NO	YES	NO
3	NO	YES	NO	NO
4	NO	YES	NO	NO
5	NO	YES	NO	NO
6	YES	NO	NO	NO
7	NO	YES	NO	NO
8	NO	YES	NO	NO
9	NO	YES	NO	NO
10	NO	YES	NO	NO
11	NO	YES	NO	NO
12	YES	NO	NO	NO
13	YES	NO	NO	NO
14	YES	NO	NO	NO
15	NO	YES	NO	NO
16	NO	NO	YES	NO
17	NO	NO	YES	NO
18	NO	NO	NO	YES
19	NO	NO	NO	YES
20	NO	NO	NO	YES
21	NO	NO	YES	NO
22	NO	YES	NO	NO
23	NO	YES	NO	NO
24	NO	YES	NO	NO
25	NO	YES	NO	NO

CLONIDINE

S. NO	NOT SATISFIED	SATISFIED	GOOD	EXCELLENT
1	YES	NO	NO	NO
2	YES	NO	NO	NO
3	YES	NO	NO	NO
4	NO	YES	NO	NO
5	YES	NO	NO	NO
6	YES	NO	NO	NO
7	YES	NO	NO	NO
8	YES	NO	NO	NO
9	YES	NO	NO	NO
10	NO	NO	YES	NO
11	NO	NO	YES	NO
12	YES	NO	NO	NO
13	YES	NO	NO	NO
14	YES	NO	NO	NO
15	YES	NO	NO	NO
16	YES	NO	NO	NO
17	YES	NO	NO	NO
18	YES	NO	NO	NO
19	YES	NO	NO	NO
20	YES	NO	NO	NO
21	NO	YES	NO	NO
22	NO	YES	NO	NO
23	YES	NO	NO	NO
24	NO	NO	NO	NO
25	NO	YES	NO	NO

RAMSAY SEDATION SCORE

DEXMEDETOMIDINE

S NO	0 MIN	60 MIN	120 MIN	180 MIN	240 MIN	300 MIN	360 MIN
1	3	3	3	3	3	3	2
2	3	3	3	3	3	3	3
3	3	3	3	3	3	3	3
4	3	3	3	3	3	3	3
5	3	3	3	3	3	3	3
6	3	3	3	3	2	2	2
7	3	3	3	3	3	3	3
8	3	3	3	3	3	3	3
9	3	3	3	3	3	3	3
10	3	3	3	3	3	3	3
11	3	3	3	3	3	3	3
12	3	3	3	3	2	2	2
13	3	3	3	3	2	2	2
14	3	3	3	3	2	2	2
15	3	3	3	3	3	3	2
16	3	3	3	3	3	3	3
17	3	3	3	3	3	3	2
18	3	3	3	3	3	3	2
19	3	3	3	3	3	3	2
20	3	3	3	3	3	3	2
21	3	3	3	3	3	3	2
22	3	3	3	3	3	3	3
23	3	3	3	3	3	3	3
24	3	3	3	3	3	3	3
25	3	3	3	3	3	3	3

CLONIDINE

S NO	0 MIN	60 MIN	120 MIN	180 MIN	240 MIN	300 MIN	360 MIN
1	3	3	3	3	2	2	2
2	3	3	2	2	2	2	2
3	3	3	3	3	2	2	2
4	3	3	3	3	2	2	2
5	3	3	3	3	2	2	2
6	3	3	3	3	2	2	2
7	3	3	2	2	2	2	2
8	3	3	2	2	2	2	2
9	3	3	3	3	2	2	2
10	3	3	3	3	2	2	2
11	3	3	3	3	2	2	2
12	3	3	2	2	2	2	2
13	3	3	3	3	2	2	2
14	3	3	3	3	2	2	2
15	3	3	3	3	2	2	2
16	3	3	3	3	2	2	2
17	3	3	3	3	2	2	2
18	3	3	3	3	2	2	2
19	3	3	2	2	2	2	2
20	3	3	3	3	2	2	2
21	3	3	3	3	2	2	2
22	3	3	3	3	2	2	2
23	3	3	2	2	2	2	2
24	3	3	3	3	2	2	2
25	3	3	3	3	2	2	2

SIDE EFFECTS – DEXMEDETOMIDINE

S.NO	HYPOTENSION	BRADYCARDIA	NAUSEA	VOMITING	DIZZINESS	DRY MOUTH	RD
1	NO	NO	NO	NO	NO	NO	NO
2	NO	NO	NO	NO	NO	NO	NO
3	NO	NO	NO	NO	NO	NO	NO
4	NO	NO	NO	NO	NO	NO	NO
5	NO	NO	NO	NO	NO	NO	NO
6	NO	YES	NO	NO	NO	NO	NO
7	NO	NO	NO	NO	NO	NO	NO
8	NO	NO	NO	NO	NO	NO	NO
9	NO	NO	NO	NO	NO	NO	NO
10	YES	NO	NO	NO	YES	NO	NO
11	NO	NO	NO	NO	NO	NO	NO
12	NO	NO	NO	NO	NO	NO	NO
13	NO	NO	NO	NO	NO	NO	NO
14	NO	NO	NO	NO	NO	NO	NO
15	NO	NO	NO	NO	NO	NO	NO
16	YES	YES	NO	NO	YES	NO	NO
17	NO	NO	NO	NO	NO	NO	NO
18	NO	NO	NO	NO	NO	NO	NO
19	NO	NO	NO	NO	NO	NO	NO
20	NO	NO	NO	NO	NO	NO	NO
21	NO	NO	NO	NO	NO	NO	NO
22	NO	NO	NO	NO	NO	NO	NO
23	NO	NO	NO	NO	NO	NO	NO
24	NO	NO	NO	NO	NO	NO	NO
25	NO	NO	NO	NO	NO	NO	NO

SIDE EFFECTS – CLONIDINE

S.NO	HYPOTENSION	BRADYCARDIA	NAUSEA	VOMITING	DIZZINESS	DRY MOUTH	RD
1	YES	NO	NO	NO	YES	NO	NO
2	NO	YES	NO	NO	NO	NO	NO
3	YES	NO	YES	YES	NO	NO	NO
4	NO	NO	NO	NO	NO	NO	NO
5	NO	NO	NO	NO	NO	NO	NO
6	NO	NO	NO	NO	NO	NO	NO
7	YES	NO	NO	NO	YES	NO	NO
8	NO	NO	NO	NO	NO	NO	NO
9	NO	NO	NO	NO	NO	NO	NO
10	NO	NO	NO	NO	NO	NO	NO
11	NO	NO	NO	NO	NO	NO	NO
12	NO	NO	NO	NO	NO	NO	NO
13	NO	NO	NO	NO	NO	NO	NO
14	NO	NO	NO	NO	NO	NO	NO
15	NO	NO	NO	NO	NO	NO	NO
16	NO	YES	YES	YES	NO	NO	NO
17	YES	NO	NO	NO	NO	NO	NO
18	NO	YES	NO	NO	NO	YES	NO
19	NO	YES	NO	NO	NO	YES	NO
20	YES	NO	NO	NO	YES	YES	NO
21	NO	NO	NO	NO	NO	NO	NO
22	NO	NO	NO	NO	NO	NO	NO
23	YES	NO	NO	NO	YES	NO	NO
24	NO	NO	NO	NO	NO	NO	NO
25	NO	NO	NO	NO	NO	NO	NO